A STUDY OF DEAFNESS IN TERM INFANTS WITH BIRTH ASPHYXIA BY OTOACOUSTIC EMISSIONS AND BRAIN STEM EVOKED RESPONSE AUDIOMETRY TESTS

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CERTIFICATE

This is to certify that this dissertation entitled "A STUDY OF DEAFNESS IN TERM INFANTS WITH BIRTH ASPHYXIA BY OTOACOUSTIC EMISSIONS AND BRAINSTEM EVOKED RESPONSE AUDIOMETRY TESTS" submitted by DR.C.MAHESWARAN to the faculty of OTORHINO LARYNGOLOGY, The TamilNadu Dr. M.G.R. Medical University, Chennai, in partial fulfilment of the requirement in the award of degree of M.S.Degree, Branch – IV (OTO - RHINO LARYNGOLOGY), for the March 2007 examination is a bonafide research work carried out by him under our direct supervision and guidance.

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DECLARATION

I, Dr. C. MAHESWARAN declare that the dissertation titled "A STUDY OF DEAFNESS IN TERM INFANTS WITH BIRTH ASPHYXIA BY OTOACOUSTIC EMISSIONS AND BRAINSTEM EVOKED RESPONSE AUDIOMETRY TESTS" has been prepared by me.

This is submitted to The Tamil Nadu Dr. M.G.R. Medical University, Chennai, in partial fulfilment of the requirement for the award of M.S.Degree,Branch IV(OTO - RHINO LARYNGOLOGY) degree Examination to be held in March 2007.

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CONTENTS

INTRODUCTION

There are many causes for hearing impairment in infants. Birth Asphyxia is one among them.

The agony and handicap caused by hearing impairment to a child is far beyond hearing alone, as we all know that a good hearing is essential for normal development of speech, language and cognitive functions of the child. So, early diagnosis of hearing impairment is essential for early initiation of rehabilitative measures in a child which is important for future speech, language and cognitive development.

Most of the tests used for assessing the hearing status in an individual requires the cooperation of the subjects which is obviously not possible in an infant.

In this study, we have used Oto acoustic emissions (OAE) test as a screening test, and Brain Stem evoked response audiometry test (BERA) as a definite test for hearing assessment. These tests are objective tests and does not require the patient's cooperation for testing, thus can be used effectively in infants.

AIM OF THE STUDY

1. To evaluate for hearing impairment in term birth asphyxiated hypoxic ischemic encephalopathy stage 2 infants using Oto acoustic emissions and Brainstem evoked response Audiometry (BERA) tests.
2. Hearing impairment if present – Assessing its severity, and to look for possible site of pathology : conductive, sensory (cochlear) or Neural (Retro-cochlear).
3. Early referral of hearing impaired children for rehabilitative measures.

REVIEW OF LITERATURE

White KR, Vohr BR, Maxon AB et al have published a paper in International Journal of Paediatric Otorhinolaryngology stating that transient evoked oto acoustic emissions is a promising technique for screening newborns for hearing loss and it could be used in a wide basis.

C.Yoshinaga itano, A.L. Gedey & DK coutler et al have done a study on language development in hearing impaired. Their finding was that in children in whom the hearing loss was identified early ie. by six months of age and appropriate rehabilitative measures were started, had a better language scores than those who were identified later than six months of age.

Behrens TR, Vohr BR & White KR et al have published a report quoting the usefulness of transient evoked oto acoustic emissions in universal screening of new born infants for hearing loss at Rhodes island.

Kemp DT & Ryans have published a paper quoting the use of transient evoked oto acoustic emissions in neonatal screening programme.

A similar report was also published by Johnson AJ & Maxon AB et al.

Fortnum H, Framworth A & Davis A et al have done a study on the feasibility of evoked oto acoustic emissions on inpatient hearing check after meningitis.

Dr.Owen et al from Department of pediatrics, Gloucestershire have studied the possibility of community based universal neonatal screening by health visitors using oto acoustic emissions. Health visitors were able to perform OAE in local health centers. They were able to achieve high population coverage rates.

Welzl Muller K. Boheim K. Stephank et al have published a report on optimizing hearing screening by transient evoked oto acoustic emissions in newborn infants. They have advised the following. A pass in one ear is enough not required to get pass result in both ears. Perform the testing after the second post partum day. A single testing is not enough and it is a must to perform oto acoustic emission testing atleast twice to minimize the false positive results.

Steven JC. Webb HB, Hutchinson J & Connell J et al have published a report on comparison between click evoked oto acoustic emissions and auditory Brain stem evoked response which states that the results by both tests are comparable.

Hunter M. Kimmi, Cafarelli Dees D et al have published a report stating the feasibility of oto acoustic emission detection followed by Auditory Brain stem evoked response audiometry in universal screening of neonates for hearing impairment.

Heinemann & Bohnert A have published a paper quoting the comparative studies and cost analysis with different instruments in screening for hearing impairment in children. They have suggested that a cost effective way for hearing analysis is to do oto acoustic emission testing universally for all children and then in those who fail the test, Auditory Brain Stem evoked response audiometry can be done.

Doyle KJ Burggruff B, Fujikawa S & Kim J have compared the utility of oto acoustic emission testing and auditory brainstem evoked response audiometry. Their reference is that in both the testing modalities, there is no obvious difference in test results.

Kennedt CR & Kimm et al have also published a similar report in archives of diseases of child hood.

Alex R. Kemper & Stephen M. Downs et al have done a cost effect analysis of newborn hearing screening strategies comparing the universal screening with oto acoustic emissions followed by BERA and Targeted screening of High risk infants for hearing loss in two stage process. The result of their study was that the universal screening can diagnose more cases at the expense of greater cost and more false positive screening results.

Sun JH, Li J Huang P et al from Shanghai medical university have published a report stating that critically ill neonates with some specific high risk factors had a significantly high incidence of hearing impairment and therefore early hearing screening is necessary for neonates who are discharged from neonatal intensive care unit.

Joint committee on infant hearing have given some guidelines for early detection of hearing impairment. They have advised hearing screening for infants with.

1. Family History of hereditary childhood sensorineural hearing loss.
2. In utero infections (TORCH, Syphilis).
3. Craniofacial anomalies involving pinna and ear canal.
4. Birth weight less than 1.5 kg.
5. APGAR scores of 0 to 4 at one minute & 0 to 6 at 5 minutes.
6. Mechanical ventilation lasting 5 days or more.
7. Hyperbilirubinemia requiring exchange transfusion.
8. Ototoxic medications – multiple courses of aminoglycosides and loop diuretics.
9. Stigmata associated with a syndrome known to include hearing loss.
10. Head trauma associated with loss of consciousness or skull fractures.
11. Bacterial meningitis.
12. Recurrent or persistent otitis media with effusion for atleast 3 months.
13. Parental concern regarding hearing or development delay.

American Academy of pediatrics, Task force on newborn infant hearing loss detection and intervention has also proposed similar guidelines for hearing screening.

Christione Mayer, Jon Witte, Agner Hildman et al have published a report on neonatal screening for hearing impairment, in which they have considered some other factors also apart from what is stated by joint committee on infant hearing. They have analysed the relation between hearing loss and maternal drug abuse, persistent pulmonary hypertension in neonate, intracranial hemorrhage of Grade III and above and periventricular leucomalacia and they have found to have a positive correlation.

Cone Wesson, Barbara Betty & Rsinger et al in their study on identification of neonatal hearing screening have stated that it is essential to do a universal screening rather than a selective high risk screening.

Wessex universal neonatal hearing trial group have also advised universal screening to prevent permanent childhood hearing impairment and its handicaps.

ANATOMY OF HEARING PATHWAY

Hearing is the function of ear. Among the special senses inner ear is the first to be fully formed in humans. It has been proved by various studies that human fetus is able to hear from 27th week of gestation onwards.

1. EXTERNAL EAR :

Hearing pathyway starts from external ear. It comprises of pinna, external auditory canal and the tympanic membrane.

PINNA AND EXTERNAL AUDITORY CANAL

1. PINNA

The pinna acts to focus and aid in the localization of sound. The entire pinna, excepts its lobule, and the outer part of the external acoustic meatus are made up of a single piece of yellow elastic cartilage covered with skin. The lateral surface of the pinna is dominated by concavities in particular the concha. The skin of the lateral and medial surfaces of the pinna possesses hair and both sebaceous and sudoriferous glands.

2. EXTERNAL AUDITORY CANAL : EAC

It extends from bottom of the concha to the tympanic membrane. It is about 24 mm long. The lateral 1/3 (8mm) of the External Auditory Canal comprises a continuation of the cartilage of the pinna. The medial 2/3 is Bony (16mm).

Nerve Supply : 1. Pinna

a) Greater auricular (C2, 3) Nerve

b) Lesser Occipital (C2) Nerve

c) Auriculo temporal Nerve (V)

d) Auricular branch of Vagus (Arnold's N)

2. External Auditory Canal :

Auriculotemporal nerve and auricular branch of vagus.

Vascular supply :

Two branches of external carotid artery, the posterior auricular artery and the superficial temporal artery.

TYMPANIC MEMBRANE :

Sound conducted through External Auditory Canal vibrates the tympanic membrane.

It forms a partition between the External Auditory Canal and the middle ear. Tympanic membrane can be divided into two parts.

a. Pars tensa :

It forms most of tympanic membrane. It's periphery is thickened to form a fibro cartilaginous ring called the annulus tympanicus, which fits in the tympanic sulcus. The central part of pars tensa is tented inwards at the level of the tip of malleus and is called the umbo.

b. Pars flaccida (Sharpnel's membrane)

This is situated above the lateral process of malleus between the notch of rivinus and the anterior and posterior malleolar folds.

Nerve supply :

Auriculo temporal nerve, auricular branch of vagus and tympanic br of glossopharyngeal N. (Jacobson's N)

MIDDLE EAR

MIDDLE EAR OSSICLES

Three ossicles – malleus, incus and stapes conduct sound energy from the tympanic membrane to the oval window and then to the inner ear fluid.

1. The malleus :

It has head, neck, handle, a lateral and an anterior process. Head and neck of malleus lie in the attic. The handle is embedded in the fibrous layer of the tympanic membrane. The lateral process forms a knob – like projection on the outer surface of the tympanic membrane and gives attachment to the anterior and posterior malleolar folds.

2. The incus :

It has a body and a short process both of which lie in the attic and a long process which has attached to the head of stapes.

3. The Stapes :

It has a head, neck, anterior and posterior crura and a foot plate. The foot plate is held in the oval window by annular ligament.

There are two muscles, tensor tympani and stapedius which regulate the magnitude of sound.

III – Inner Ear :

Inner ear consists of cochlea and vestibule. Vestibule contains semicircular canals, saccule and the utricle.

THE COCHLEA AND ITS NEURAL CONNECTIONS

COCHLEA :

Cochlea is buried in hardest bone of the body, the petrous part of temporal bone. Cochlea contains the receptors for hearing. Cochlea is snail shaped. It is 3 cm long and makes 2 ¾ turns around the central axis called modiolus. The canal is divided by two membranes ie. Basilar and Reisner's membrane into three compartments, the upper scala vestibuli, the middle scala media, the lower scala tympani. Scala media contains the endolymph. The scala vestibuli and scala tympani contain the perilymph. The receptors for hearing, the hair cells are located on the basilar membrane in scala media.

The hair cells are divided into inner and outer hair cells by pillars of corti. The hair cells contain stereocilia. They are in contact with tectorial membrane. Together they are called as organ of corti. The perilymphatic space of scala tympani is continuous with subarachnoid space of posterior fossa through cochlear aqueduct. This aqueduct is patent in neonatal period. This is the

reason for post bacterial meningitic bilateral deafness with vestibular impairment.

Auditory pathway :

Auditory pathway begins with auditory nerve endings at base of hair cells. The cell bodies of which are in spiral ganglion in Rossenthals canals.

On entering the brain stem, auditory fibres bifurcate into upper division and the lower division. The upper division ends in dorsal cochlear nuclei on both sides. So they form a cross over in the midline. This cross over forms the acoustic striae. The lower division ends in ventral cochlear nucleus. Second order neurons from the ventral cochlear nucleus ends in superior olivary nucleus on both sides. This cross over is called by the name trapezoid body.

Second order neurons from dorsal cochlear nucleus ascends in lateral lemnicus to relay at the inferior cochlear nucleus.

Similarly fibres ascending from superior olivary nucleus ascend in lateral lemniscus and end in inferior colliculus on enroute some fibres relay in nucleus of lateral lemniscus. There is a cross

over between fibres of nucleus of lateral lemniscus of both sides which terms the commissure of probst.

There is a cross over of fibres between inferior colliculus on both sides which forms the inter collicular commissure.

From the inferior colliculus the fibres ascend to the medial Geniculate body. From the medial geniculate body fibres are projected to the Auditory Cortex (area 41 & 42)

Area 41 the Heschl's gyrus is the primary auditory area where pitch and intensity discrimination occurs. Area 42 is auditory association area where complex synthesis of sound occurs. In auditory area of brain there is cochleotopic representation as if cochlea is unwinded on cortex with apex represented on outer aspect and base of cochlea on inner aspect.

The two lateral lemniscus and four cross over ie. Trapezoid body, Acoustic striae, commissure of probst and inter collicular fibres forms a ladder pattern.

PHYSIOLOGY OF HEARING MECHANISM

Any vibrating object causes waves of compression and rarefaction and is capable of producing sound. In the air, at 20^{o}C and at sea level, sound travels at a speed of 344 metres (1120 feet) per second. It travels faster in liquids and solids than in the air. Also, when sound energy has to pass from air to liquid medium, most of it is reflected because for the impedance offered by the liquid.

Mechanism of hearing :

A sound signal in the environment is collected by the pinna, passes through external auditory canal and strikes the tympanic membrane. Vibrations of the tympanic membrane are transmitted to stapes footplate through a chain of ossicles coupled to the tympanic membrane. Movements of stapes footplate cause pressure changes in the labyrinthine fluids which move the basilar membrane. This stimulates the organ of corti. It is these hair cells which act as transducers and convert the mechanical energy into electrical impulses which travel along the auditory nerve. Thus, the mechanism of hearing can be broadly divided into :

1. Mechanical conduction of sound (conductive apparatus)
2. Transduction of mechanical energy to electrical impulses (sensory system of cochlea)
3. Conduction of electrical impulses to the brain (neural pathways)

1. Conduction of sound :

A person under water cannot hear any sound made in the air because 99.9% of the sound energy is reflected away from the surface of water because of the impedance offered by it. A similar situation exists in the ear when air conducted sound has to travel to cochlear fluids. Nature has compensated for this loss of sound energy by interposing the middle ear which converts sound of greater amplitude, but lesser force, to that of lesser amplitude and greater force. This function of the middle ear is called impedance matching mechanism or the transformer action.

It is accomplished by :

a) Lever action of the ossicles :

Handle of malleus is 1.3 times longer than long process of the incus, providing a mechanical advantage of 1.3.

b) Hydraulic action of tympanic membrane :

The area of tympanic membrane is much larger than the area of stapes foot plate, the average ratio between the two being 21:1. As the effective vibratory area of tympanic membrane is only two-thirds, the effective areal ratio is reduced to 14:1 and this is the mechanical advantage provide by the tympanic membrane.

The product of areal ratio and lever action of ossicles is 18 : 1

C. Curved membrane effect :

Movements of tympanic membrane are more at the periphery than at the centre where malleus handle is attached. This too provides some leverage.

PHASE DIFFERENTIAL BETWEEN OVAL AND ROUND WINOW :

Sound waves striking the tympanic membrane do not reach the round and oval windows simultaneously. There is a preferential pathway to the oval window because of the ossicular chain. Thus, when oval window is receiving wave of compression, the round window is at the phase of rarefaction. If the sound waves were to strike both the windows simultaneously, they would cancel each

other effect with no movement of the perilymph and no hearing. This acoustic separations of windows is achieved by the presence of intact tympanic membrane and a cushion of air in the middle ear around the round window.

NATURAL RESONANCE OF EXTERNAL AND MIDDLE EAR :

Inherent anatomic and physiologic properties of the external and middle ear allow certain frequencies of sound to pass more easily to the inner ear due to their natural resonances. Natural resonance of external ear canal is 300 Hz. Frequencies most efficiently transmitted by ossicular chain are between 500 and 2000 Hz while that by tympanic membrane is 800-1600 Hz. Thus greatest sensitivity of the sound transmission is between 500 and 3000Hz and these are the frequencies most important to man in day to day conversation.

2. TRANSDUCTION OF MECHANICAL ENERGY TO ELECTRICAL IMPULSES :

Movements of the stapes foot plate, transmitted to the cochlear fluids, move the basilar membrane, setting up shearing force between the tectorial membrane and the hair cells. The distortion of

hair cells gives rise to cochlear microphonics which trigger the nerve impulse.

A sound wave, depending on its frequency, reaches maximum amplitude on a particular place on the basilar membrane and stimulates that segment. Higher frequencies are represented in the basal turn of the cochlea and a progressively lower ones towards the apex.

3. Neural pathways :

Hair cells get innervation from the bipolar cells of spiral ganglion. Central axons of these cells collect to form cochlear nerve which goes to ventral and dorsal cochlear nuclei. From there, both crossed and uncrossed fibers travel to the superior olivary nucleus, lateral lemniscus, inferior colliculus, medial geniculate body and finally reach the auditory cortex of the temporal bone.

ETIOLOGY OF HEARING LOSS IN INFANTS

Etiology of hearing loss :

Hearing loss can be central or peripheral in origin. The peripheral hearing loss is further divided into

1. Conductive hearing loss
2. Sensori neural hearing loss
3. Mixed hearing loss

1. Conductive hearing loss :

This is commonly caused by dysfunction in the transmission of sound through the external or middle ear. It may be congenital or acquired.

A. Congenital :

i) Anomalies of pinna, external ear canal, tympanic membrane and ossicles. (Most common cause of congenital conductive hearing loss)

ii) Genetic conditions

a) Pierre Robin syndrome

b) Treacher Collins syndrome

c) Klippel feil syndrome

d) Crouzon's syndrome

iii) Congenital cholesteatoma (very rarely)

B. Acquired

i) Otitis media both acute & chronic variety and its complications like effusion, cholesteatoma, tympanosclerosis & adhesive otitis.

ii) Impacted wax or cerumen

iii) Impacted foreign body

iv) Tympanic membrane perforation (due to trauma or otitis media)

v) Otosclerosis

vi) Osteogenesis imperfecta

vii) Osteopetrosis

viii) Tumors in the ear canal or middle ear (Osteomas, eosinophilic granuloma, rhabdomyosarcoma)

2. Sensorineural hearing loss :

It is the type of hearing loss where the inner ear or the eighth cranial nerve is involved resulting in impairment of sound

perception in the cochlea and higher centres. Sensorineural hearing loss can be because of congenital or acquired causes.

A. Congenital :

i) Genetic causes :

a) Autosomal Recessive syndromes

a) Usher's syndrome

b) Pendred's syndrome

c) Jervell-Lange Nielsen syndrome (a form of the long Q.T. interval syndrome)

b) Autosomal Dominant

a) Waardenburg's syndrome

b) Brachio-otorenal syndrome

c) Sex linked syndrome

a) Alport's syndrome

b) Norrie's syndrome

d) Chromosomal Abnormalities :

a) Down's syndrome

b) Turner's syndrome

c) Trisomy 18 & 13

ii) Infection (intrauterine infections)

a. Rubella

b. Cytomegalovirus

c. Toxoplasmosis

d. Syphilis

iii) Teratogenic

a. Thalidomide

b. Quinine

c. Aminoglycosides

d. Loop diuretics

e. Cisplatin

B. Acquired :

1. Perinatal asphyxia – very important cause of hearing loss in infants in the absence of any congenital causes of hearing loss.

2. Kernicterus

3. Prematurity

4. Infections

a) Bacterial Meningitis

i) Pneumococcus

ii) Hemophilus influenza

iii) Meningococcus

b) Viral infections

i) Measles

ii) Mumps

iii) Rubella

iv) Varicella

5. Ototoxic drugs :

i) Quinine

ii) Aminoglycosides

iii) Loop diuretics

iv) Cisplatin

v) Salicylates

6. Traumatic causes

i) Fracture temporal bone

ii) Head injury

iii) Barotrauma

iv) Noise (acoustic trauma)

Central Causes :

Auditory deficits originating along the central auditory nervous system pathways from the proximal eighth nerve to the cerebral cortex are generally considered central hearing loss.

HEAD TO FOOT EXAMINATION OF A CASE WITH HEARING LOSS

1. Face and head

Look for any abnormalities in shape, symmetry & presence of any skin tags.

2. Eyes :

Look for intercanthal distance, slant, it's colour, vision and retina.

3. Ears

- Downward slanting palpebral fissures, coloboma of lower eyelid, malar hypoplasia, malformation of external ear with or without atresia of ear canal, preauricular skin tags, dental malocclusion, teeth hypoplasia & cleft palate are features of Treacher Collins syndrome.

- Anterior lenticonus is present in Alport's syndrome.
- Myopia, cataract, retinal detachment, arthropathy, cleft palate and micrognathia in sticklers syndrome.
- Retinitis pigmentosa is present in Usher's syndrome.
- Bilateral acoustic neuroma caféaulait spots and sub capsular cataract occur in Neuro fibromatosis type – 2.

4. Hair

Look for texture, colour & white forelock.

White forelock, premature graying of hair, heterochromia iridis, hypertension and partial albinism are features of waardenburg's syndrome.

5. Neck

- Look for sinus tracts, thyromegaly.
- Thyroid enlargement can occur in Pendred's syndrome.
- Branchial clefts, fistula and cysts with malformed pinna preauricular pits & renal anomalies occur in Branchio otorenal syndrome.

6. Skin

Look for cafeaulait spots, hypopigmentation, hyper-pigmentation and axillary freckling - can occur with Neurofibromatosis type I.

7. Balance & gait

Gait disturbance can occur in Usher's syndrome due to vestibular dysfunction.

EXAMINATION OF EAR / NOSE AND THROAT

EAR

1. Pre auricular area :

   Look for any pre auricular sinus, skin tags, and pits.

2. Pinna :

   Its size and shape (Anotia, microtia, or macrotia).

3. Post auricular area :

   To look for any lymphnodes, malformation etc.

4. EAC (External auditory canal) :

   Its size and shape – to look for wax, foreign body, vernix caseosa, congenital aplasia, hypoplasias.

5. Otoscopy & Otomicroscopy :

To assess the color, mobility of tympanic membrane.

To look for any perforation , fluid level, retraction.

6. Facial Nerve :

Facial Palsy could be idiopathic or during delivery.

THROAT

1. Oral Cavity and Oropharynx :

To rule out cleft lip, cleft palate. To look for dental hypoplasia.

Nose :

To assess position of septum, Mucosa, turbinates.

BIRTH ASPHYXIA

Asphyxia refers to a comibination of hypoxia, hypercarbia and metabolic acidosis. National neonatology forum of India has suggested that birth Asphyxia should be diagnosed when "baby has gasping and inadequate breathing or no breathing at 1 minute". It corresponds to 1 minute apgar score of 3 or less.

The American academy of pediatrics has proposed that the term perinatal asphyxia should be reserved to describe an infant who manifests with all of the following features :

1. Cord umbilical artery PH of < 7.0 with a base deficit of > 10m Eq / litre.
2. Neonatal neurologic manifestations suggestive of Hypoxic ischemic encephalopathy.
3. Evidences of multisystem organ dysfunction (eg. Cardio vascular, renal, Gastrointestinal, hematologic or pulmonary).

ETIOLOGY :

1. Meconium aspiration or tracheal plug.
2. Congenital malformations (Choanal atresia, laryngeal web, diaphragamatic hernia, Oesophageal atresia with tracheo oesophageal fistula, lobar emphysema or cyst).
3. Pneumothorax and pneumomediastinum.
4. Intracranial hemorrhage.
5. Shock (Cardiogenic or hypovolemic).
6. Hydrops fetalis.
7. Immaturity.
8. Paralysis of respiratory muscles.
9. Pulmonary hemorrhage.
10. Excessive maternal sedation.

PATHOPHYSIOLOGY:

Birth asphyxia is associated with reduction in the arterial oxygen tension, accumulation of CO2 and fall in blood pH. Acidosis occurs due to anaerobic utilization of glucose, production of lactic acid and accumulation of CO2. These biochemical changes cause

constriction of relatively muscular pulmonary arterioles and raise the pulmonary arterial pressure. This results in reduced filling of the left heart and right to left shunts. Severe and prolonged hypoxia leads to depletion of cardiac glycogen stores and tendency towards hypoglycemia.

Hypothermia and hypoglycemia lead to accumulation of non esterified fatty acids and glycerol. Due to intracellular hypoxia, net catabolism of ATP results in release of adenine metabolities. Anoxic damage to cells result in failure of energy dependent sodium pump mechanism with release of potassium and phosphates into the extracellular fluid. Petechial hemorrhages due to anoxic capillary damage, intracellular collection of sodium and inappropriate release of ADH are associated with development of cerebral edema.

SYSTEMIC MANIFESTATIONS OF BIRTH ASPHYXIA:

1. Brain : Hypoxic Ischemic encephalopathy, intracranial hemorrhage, seizures, sensorineural hearing loss, apnoeic attacks.
2. Heart : Dysrrhythmias, myocardial damage, CCF.

3. Lungs : Meconium aspiration, hyaline membrane disease, pulmonary hemorrhage, pnemonia, pneumothorax, shock lung.

4. Kidneys : Hematuria, renal failure, acute tubular necrosis, renal vein thrombosis.

5. Hematologic : Coagulopathy (Disseminated intra vascular coagulopathy) ; Sepsis.

6. Gastro Intestinal : Necrotising enterocolitis, paralytic ileus.

7. Endocrine : SIADH, Adrenal Hemorrhage.

8. Immunologic : Septicemia.

Hypoxic Ischemic Encephalopathy:

Neonatal encephalopathy following severe birth asphyxia or perinatal hypoxia is referred to Hypoxic ischemic encephalopathy (HIE). Cerebral ischemia occurs as a consequence of cerebral edema and reduced cerebral perfusion due to myocardial dysfunction as a result of hypoxic cardiomyopathy. Following severe birth asphyxia 25 percent infants are likely to develop the syndrome of HIE.

S.No.	Features	Stage 1	Stage II	Stage III
1.	Consciousness	alert	lethargic	Comatosed
2.	Muscle tone	normal	hypotonic	Flaccid
3.	Tendon reflexes	Brisk	Exaggerated	Absent
4.	Myoclonus	present	present	Absent
5.	Sucking	Active	Weak	Absent
6.	Mororesponse	Exaggerated	incomplete	Absent
7.	Grasping	Normal	Exaggerated	Absent
8.	Oculocephalic reflex	Normal	Over reactive	Absent
9.	Pupils	Dilated	constricted	dilated& fixed
10.	Respiration	Regular	Periodic	Apnoeic attack
11.	Heart Rate	Normal	bradycardia	brady cardia
12.	Seizures	Absent	Common	Uncommon
13.	EEG	Normal	Lowvoltage, periodic	Periodic Iso Electric

MANAGEMENT :

Efforts should be made to prevent further hypoxic damage to the brain and correct any associated acid – base and metabolic abnormalities. If despite active resuscitation efforts, 5 minute apgar score is less than 5, the infant should be admitted in the neonatal intensive care unit for close monitoring and management.

OTO ACOUSTIC EMISSIONS (OAE)

Oto acoustic emissions were first discovered by Dr. David Kemp in 1978. The first commercial equipment for recording OAE was produced in USA by 1988. Since then Oto acoustic emission testing is used for screening hearing impairment.

What is Oto acoustic Emission :

A disturbance in the environment causes sound waves to be created which travel through the air. The sound is funneled into the ear canal by the pinna and it strikes the tympanic membrane. Then it is transmitted through the middle ear through the ossicles malleus, incus and stapes. The foot plate of stapes conducts the traveling waves across the oval window. Thus the sound reaches the fluid filled cochlea and vibrates the basilar membrane. Each portion of basilar membrane is maximally sensitive to only a limited frequency range. The arrangement is a tonotopic gradient. Regions closest to the oval window are more sensitive to high frequency stimuli. Regions further away are more sensitive to lower frequency stimuli. On the basilar membrane lies the small receptor cells called hair cells. They are called so because their appearance resembles small

hair follicles. A closer look at hair cells show that they are arranged in rows.

The inner hair cells are arranged in single row and the outer hair cells are arranged in multiple rows. (three to four)

When the basilar membrane vibrates the hair cells are set into motion and an electro mechanical response is elicited, while an afferent signal is transmitted to the brain an efferent signal is also emitted by the outer hair cells. These efferent signals we call by the name oto acoustic emissions (OAE). The OAE travels in the reverse direction from cochlea through the ossicular chains vibrating the tympanic membrane to the external auditory canal. When we use special sensitive equipment with a probe in auditory canal these oto acoustic emissions can be recorded.

There are four types of oto acoustic emissions :

1. Spontaneous Oto acoustic emissions (SOAE)
2. Transient evoked Oto acoustic emissions (TEOAE)
3. Distortion product Oto acoustic emissions (DPOAE)
4. Sustained frequency Oto acoustic emissions (SFOAE)

Spontaneous Oto acoustic emissions :

These are sounds produced without any auditory stimuli. These non evoked response usually is measured in narrow bands (<30 Hz bandwidth) of frequencies. Obtain multiple recordings to ensure replicability and to distinguish the response from the noise floor. SOAE recordings usually span 500 to 7000 Hz frequency range.

Transient Evoked Oto Acoustic emissions :

In this an auditory stimuli is given and the OAE emitted by outer hair cells are recorded. Clicks are the most commonly used stimuli, although tone burst stimuli may be used. Most commonly 80 to 85 dB spl stimuli are used clinically. The stimulation rate is less than 60 stimuli per second. TEOAE are generally recorded in the time domain over approximately 20 milli seconds. Alternating responses are stored in alternating computer memory banks A and B. Data that correlate between the two memory banks are considered as a response. Data that do not correlate are considered noise. When present TEOAE generally occur at frequencies of 500 to 4000 Hz. Data in the time domain then are converted to the frequency domain, usually in octave band analysis.

Distortion product Oto acoustic emissions :

In this stimuli consists of two pure tones at two frequencies (f1, f2, (f2 > f1) and two intensity levels (i.e. L1 & L2). The relationship between L1-L2 and f1/f2 dictates the frequency response. An f1/f2 ratio yields the greatest DPOAE at 1.2 for low and high frequencies and of 1.3 for medium frequencies. To yield an optimal response, set intensities so that L1 equals or exceeds L2. Lowering the absolute intensity of the stimulus renders the DPOAEs more sensitive to abnormality. A setting of 65/55 dB SPL L1/L2 is frequently used. Responses are usually most robust and recorded at the emitted frequency of 2f1-f2 however, they generally are charted according to f2 because that region approximates the cochlear frequency region generating the response.

Sustained frequency oto acoustic emissions :

SFOAES are responses recorded to a continuous tone. Because the stimuli and emission overlap in the ear canal, the recording microphone detects both. Therefore interpretation depends on reading a complicated series of rippler in the recording. At present SFOAEs are not used clinically.

In clinical practice TEOAE and DPOAE are most commonly used. In our study we have used TEOAE for screening the infants.

Prerequisites for obtaining oto acoustic emissions :

1. Un obstructed outer ear canal (like wax).
2. Hermetic seal of the ear canal with the probe.
3. Optimal positioning of the probe.
4. Absence of middle ear pathology.
5. Functioning cochlear outer hair cells.
6. A quiescent patient, excessive movement or vocalization may preclude recording.
7. Relatively quiet recording environment. A sound booth is not required, but a noisy environment may preclude accurate recording.

Nonpathological problems that can cause absence of OAEs.

1. Poor probe tip placement or poor seal. Most current equipments alerts clinicians to these problems.
2. Standing waves – most current equipments alerts clinicians to standing waves.
3. Cerumen occluding the canal or blocking a probe port.

4. Debris and foreign objects in the outer ear canal.
5. Vernix caseosa in neonates. This is common immediately after birth.
6. Un cooperative patient. Usually, recordings simply are not obtained.

Other pathological problems that can cause absence of OAEs.

1. Stenosis of ear canal.
2. Otitis externa.
3. Cysts in ear canal.
4. abnormal middle ear pressure.
5. Otitis media.
6. Oto sclerosis.
7. Middle ear disarticulation.
8. Cholesteatoma.

Advantages of OAE :

1. Objective test, Does not require the cooperation of infants.
2. Less time consuming.
3. Less costly.

4. The probes are less invasive than electrodes required for electrical responses.
5. Can be done in a sleeping child.
6. Less distressing for the parents.
7. All frequencies are tested unlike Brain stem evoked response audiometry.
8. Response can be obtained even in the presence of tympanostomy tube.
9. Does not require a sound booth. Can be done in any quiet environment.
10. Child needs to be quite and still only for 2 to 5 minutes.

Disadvantages :

1. Cannot be recorded in presence of secretory otitis media.
2. Requires the child to be completely quiet without noisy breathing or sucking.
3. Can identify only hearing loss more than 30 dB.
4. Gives no indication of the severity of any hearing impairment.

BRAIN STEM EVOKED RESPONSE AUDIOMETRY (BERA) TEST

Introduction :

It was the early work of Berger (1930) on the recording of the electro encephalogram (EEG) from the human scalp and the follow up work by Davis Etal. (1939) which lead to subsequent refinements in recording specific changes in the EEG, and gave birth to BERA techniques as we now know them.

When a sound reaches the cochlea, it is converted into an electrical impulse and passes through the following pathway. Spiral ganglion in superior olivary complex in the mid brain – Lateral lemniscus – inferior colliculus – medial Geniculate body in thalamus – Auditory cortex. Passage of impulse through this pathway generates an electrical activity which can be monitored by placing a surface electrode on the vertex of the scalp. As such electrical activity in the brain elecited by a sound stimulus is called auditory evoked potentials (AEP). It can be recorded upto 500 milliseconds from the time of onset of sound stimulus. The AEP recorded in the first 10 milliseconds is called short latency response (SLR) and popularly known as BERA.

METHOD OF RECORDING BERA :

The recording always done after adequate sedation of child. The sedatives that can be used are syrups of chloral hydrate or promethazine orally. The auditory evoked potential is elicited by a click stimulus having an intensity of approx. 60 db above the average puretone hearing level of the subject and is recorded with the active electrode placed over the vertex. Of the other electrodes one called reference electrode is placed on the mastoid of the ipsilateral ear, and the ground electrode is placed either over forehead just above the nasion or over the contralateral mastoid.

The sound stimulus is a broad band click of 0.1 milli second duration. The click sound is fed into the ear to be tested by a head phone and the contralateral ear is suitably masked by a white noise. Stimulus rate is 33.3/sec. The lowest frequency of sound in the broad band click is 100 hz and highest frequency is about 4000 to 5000 hz.

The recording is obtained as a graph with amplitude (in microvolts) on the ordinate and time (in milliseconds) on the abscissca.

INTERPRETATION OF BERA RESPONSE :

A normal BERA recording has five prominent peaks and two small peaks. They are termed as BERA potentials or BERA waves. The waves are numbered from I to VII. Each of these waves give us information about a specific segment of the auditory pathway from the cochlea to the midbrain region.

Wave	Site of Neural Generator
I	Cochlear Nerve (Distal end)
II	Cochlear Nerve (Proximal end)
III	Cochlear Nucleus
IV	Superior Olivary complex
V	? Lateral Lemiscus ? Inferior Colliculus
VI & VII	Neural Generators not definitely known

In all of these waves, wave V is the most reliable and easily identifiable wave in tracing. Each wave has a latency period of about 1 milli second. Identitying wave I is important because it gives an idea whether the stimulus has crossed over from cochlea and the distal end of the auditory nerve.

The Parameters of the BERA that are studied are as follows :

a) Latency of the wave (s) :

i) Absolute latency : the time interval between the onset of the stimulus and the peak of the wave in milliseconds.

ii) Interwave latency : Time interval in milliseconds between two different waves in the same ear.

iii) Interaural latency diffence : The time interval between the two ears of the same wave.

b) Amplitude of the wave(s)

c) Wave form morphology

d) Latency – Intensity function of wave – V

S. No	Parameter	Normal value	Criteria for Abnormality(ms)
1.	I to III IPL	2	> 2.4
2.	III to V IPL	2	> 2.4
3.	I to V IPL	4	> 4.4
4.	Interaural latency difference of wave V	< 0.3	> 0.3
5.	Morphology of wave V	Present	Absent

CLINICAL USES OF BERA:

1. Estimation of hearing threshold :

Objective estimation of hearing threshold by BERA is very useful in newborns, infants and other difficult to test subjects who can not cooperate adequately during the behaviour tests. The BERA threshold is defined as the lowest intensity level at which a detectable and repeatable response is observed in the BERA Tracing. The degree of hearing impairment is usually assessed by gradually decreasing the intensity of the sound stimulus and noting the morphology of the graph until a time comes when the wave V is no longer identifiable. Pure tone hearing threshold may be identified by subtracting 10 db from the point at which the wave V was just identifiable.

Identification of the nature of deafness :

The latency of wave V is recorded for sound stimuli of different intensites and plotted graphically with, intensity in decibels on the ordinate and latency in milliseconds of wave V on the abscissa. In purely conductive deafness graph shifts to the right and parallel to that of normal person. In sensory (Cochlear) deafness the

graph shows a steep high sloping configuration. In neural deafness a shallow graph is obtained. The neural (Retrocochlear) deafness can also be identified by if inter aural latency difference of wave V is more than 0.3 milli sec.

Advantages of BERA :

a) Test is objective. The test can be reliably performed even when the child is sleeping or is sedated.

b) Individual ears can be tested separately.

Limitations of BERA :

a) The test is not frequency specific. Low frequency deafness is missed by this method.

b) There are some patient related variables like age and sex of the child which alter the wave characters.

c) Muscular contractions caused by the movements of the child sometimes cause artifacts which contaminate the BERA tracing.

d) The test is very time consuming. Test sessions of more than 1 hour may be required if the child is restless.

MATERIALS AND METHODS

Study Design :

Prospective longitudinal study.

Study population :

Term birth asphyxiated hypoxic ischemic encephalopathy stage 2 infants attending the well baby clinic in Institute of child health and Research Centre in Government Rajaji Hospital attached at Madurai Medical College, and referred to our department of ENT and Head and Neck surgery for hearing assessment.

Study period :

From January 2005 to June 2006.

Inclusion Criteria :

1. Term babies.
2. Birth asphysiated Hypoxic ischemic encephalopathy stage 2 infants.
3. With normal developmental milestones.
4. Without severe neurologic impairment.

Exclusion Criteria :

1. Preterm babies.
2. Babies with severe neurologic impairment.
3. Babies with other risk factors like hyperbilirubinemia.
4. Babies with other congenital anomalies.
5. Babies with family history of hearing loss.
6. Very low birth weight babies.

Method :

Term birth asphyxiated infants who are on regular follow up are initially screened for hearing by response to turning to ring of a Bell at around 6 months of age. The six month cut off is taken because the average time when a child turns to sound is around 5.8 months according to Trivandrum developmental screening test. Those children who have doubtful turning to sound by ring of a bell are subjected to oto acoustic emission test after parental consent which is an objective test for hearing impairment. Those who failed in the OAE test are further subjected to BERA test.

RESULTS AND OBSERVATIONS

TABLE – 1

Follow up of children enrolled

	Children	
	Number	%
Children followed up	176	62.4
Children lost to follow up	106	37.6
Total children enrolled	282	100

Out of the 282 children enrolled for the study, 37.6% of the children were lost to follow up due to various reasons.

TABLE –2

BELL TEST

Bell test result	Children	
	Number	%
Children Found normal	128	72.7
Children suspected to be defective	48	27.3
Total children followed up	176	100

Among the 176 children followed up, 48 (27.3%) children were suspected of having defect in the Bell test.

Table – 3

Bell test and OAE test

OAE Result	Children	
	Number	%
Children confirmed defective as per OAE Bilaterally	8	16.7
Children confirmed normal as per OAE	40	83.3
Total children suspected of having hearing defect as per Bell test	48	100

OAE test confirmed hearing defect in 16.7% of the cases among children suspected of having hearing defect in Bell test.

BERA TEST

BERA test was done only in those children confirmed defective Bilaterally as per OAE.

Table - 4

BERA Test and OAE Test

BERA TEST	Children	
	Number	%
Children with severe hearing loss	6	75
Children moderate hearing loss	2	25
Total children confirmed defective as per OAE bilaterally	8	100

In all cases site of pathology could probably be cochlea.

Table – 5

Sex wise distribution

	Hearing impairment present		Hearing impairment absent	
	No.	%	No.	%
Male	6	17.1	29	82.9
Female	2	15.4	11	84.6
Total	8	20	40	80

P = 0.6266

The percentage of deafness was slightly higher in males,

Male, female ratio is 3 :1

But there was no statistically significant difference in incidence of hearing defects among birth asphyxiated male & female babies.

Table – 6

Obstetric History

Obstetric History	Hearing impairment present		Hearing impairment absent	
	No.	%	No.	%
B.O.H. (n=10)	2	20	8	80
Normal (n=38)	6	15.8	32	84.2
Total (n=48)	8	20	40	80

P = 0.5359

The percentage of hearing defect was slightly more among those with previous bad obstetric history. But it was statistically not significant.

Table – 7

Type of Delivery

Type of Delivery	Hearing impairment present		Hearing impairment absent	
	No.	%	No.	%
Normal Delivery (n=32)	4	12.5	28	87.5
Assisted Delivery (n=16)	4	25	12	75
Total (n=48)	8	20	40	80

P = 0.2424

The percentage of hearing loss among birth asphyxiated infants delivered by assisted delivery was twice that of those delivered by labour natural.

Table : 8 Apgar Score

Apgar score 1'	Hearing impairment present		Hearing impairment absent	
	No.	%	No.	%
< 4 (n=11)	4	36.4	7	63.6
> 4 (n=37)	4	10.8	33	89.2
Total (n=38)	8	20	40	80

P = 0.0482

Apgar score 5'	Hearing impairment present		Hearing impairment absent	
	No.	%	No.	%
< 4 (n=10)	4	10	6	60
> 4 (n=38)	4	10.5	34	89.5
Total (n=48)	8	20	40	80

P = 0.0471

There is a significant correlation between APGAR Score and hearing defect. When the APGAR score was less than 4 of 1 or 5 minutes. (ie. those with severe birth asphyxia) incidence of hearing defects increases significantly.

Table – 9

Birth Weight

Birth Weight	Hearing impairment present		Hearing impairment absent	
	No.	%	No.	%
< 2.5 kg (n=6)	1	16.7	5	83.3
2.5 – 3.0 kg (n=34)	6	17.6	28	82.4
> 3.0 kg (n=8)	1	12.5	7	87.5
Total (n=38)	8	20	40	80

P = 0.6872

Very low birth weight infants have been excluded from the study. In the study group there was no obvious difference in incidence of hearing defect in various weight groups.

Table – 10

Neurosonogram / Computed Axial Tomogram Brain

Results

Neuro Sonogram / CT brain Result	Hearing impairment present		Hearing impairment absent	
	No.	%	No.	%
Normal (n=36)	5	13.9	31	86.1
Abnormal (n=12)	3	25	9	75.0
Total	8	20	40	80

P = 0.3137

The percentage of hearing loss was high among those with abnormal neurosonogram, computed axial tomogram finding when compared to those with normal findings.

Table – 11

Duration of Hospitalisation

Duration of Hospitalisation (in days)	Hearing impairment present		Hearing impairment absent	
	No.	%	No.	%
< 5 days (n=5)	-	-	5	100
5-1 (n=28)	4	14.3	24	65.7
> 10 (n=15)	4	26.7	11	73.3
Total (n=38)	8	20	40	80

P = 0.4034

In those with less than 5 days hospitalization there were no hearing defect & the percentage of hearing defect was more in those with more than 10 days hospitalization than those with less than 10 days hospitalization.

Table – 12

Socio economic status

Socio economic status	Hearing impairment present		Hearing impairment absent	
	No.	%	No.	%
I (n=0)	-	-	-	-
II (n=0)	-	-	-	-
III (n=9)	-	-	9	100
IV (n=14)	2	14.3	12	85.7
V (n=25)	6	24	19	76
Total (n=48)	8	20	40	80

P = 0.1509

The percentage of hearing defect was more in those with class V socio economic status than those with class IV socio economic status. No cases was reported in class III socio economic status.

DISCUSSION

In our study we have screened all term birth asphyxiated Hypoxic Ischemic Encephalopathy stage II infants for hearing loss using a bell and if they are found to have doubtful turning to sound in bell test they were subjected to Oto acoustic emission testing. Those who failed in the OAE test, bilaterally are further tested by BERA test. Since the mean age of turning to sound is around 5.8 months we have taken 6 months as cut off point and screened all infants at 6^{th} month while they are on follow up.

A total of 282 cases of term birth asphyxiated Hypoxic Ischemic Encephalopathy stage II infants were registered for study and out of which 106 cases were lost to follow up for various reasons. Of the remaining 176 cases who were on regular follow up 48 infants had doubtful turning to sound when they were tested by bell method. Of these 48 cases, 35 were males, 13 were females. These 48 cases were subjected to screening by oto acoustic emission testing.

Of the 48 cases tested by oto acoustic emissions 40 infants passed the test and the remaining 8 cases did not pass the test. In

BERA testing of the 8 cases, 6 of them had bilateral severe hearing impairement (75%). Two of them had bilateral moderate hearing impairement (25%). In all cases the propable site of pathology could be cochlea.

Of these 8 cases 6 were males and 2 were females. But there is no statistically significant difference in incidence of hearing loss in birth asphyxiated infants in both sexes.

Of the 8 cases mothers of 2 cases had previous bad obstetric history and in the remaining 6 cases the obstetric history was normal. So previous bad obstetric history does not affect the outcome of hearing significantly.

When comparing the hearing outcome in various mode of delivery we could find that the percentage of infants with hearing impairment in those with assisted delivery was twice as compared to babies delivered by labour natural. But the confounding factor here in that in cases which required assisted delivery already they were in a state of prolonged labour which may itself contribute to perinatal asphyxia.

Very low birth weight infants have been excluded from the study. In this study group were the birth weight ranged from 2.0 kg to 3.5 kg there was no significant difference in incidence of hearing defect in any particular weight categories of infants.

At this study was conducted in a Government hospital settings only cases belonging to class III, class IV & class V socio economic status scaling of Kuppuswamy who have utilized the hospital services have been included in the study. So the incidence of hearing impairment could not be assessed in all social classes. But among this cases no infant was found to be hearing impaired in class III and the percentage of hearing impairment was slightly higher in those belonging to class V when compared to class IV socio economic strata. But a conclusion cannot be reached on this point as this is not a population based study and most of the cases attending the government hospital belonged to lower socio economic strata.

There was a significant correlation between APGAR score and the incidence of hearing impairment. The incidence of hearing impairment was significantly higher in those infants with severe birth asphyxia i.e. infants with 5 minute APGAR score of less than 4

when compared with those of APGAR score of more than 4 at 5 minutes. So the incidence of hearing impairment is directly proportional to the severity of asphyxia.

The percentage of infants with hearing loss was higher in those with abnormal findings in neurosonogram or computed axial tomogram brain when compared to those with normal neurosonogram or computed axial tomogram Brain findings but the P value was not significant. So, this abnormal neuro imaging finding can not be taken as a positive collaborative evidence.

When considering the duration of hospitalization and number of infants with hearing loss the following observations were made. There was no infant with hearing impairment in the group of infants with less than five days of hospitalization. But the percentage of infants with hearing impairment was twice in those group of infants who required more than 10 days of hospitalization when compared to those group of infants with less than 10 days of hospitalization. So it is obvious that those infants with prolonged convulsions who required longer duration of hospital stay to control convulsions had greater incidence of hearing impairment.

LIMITATIONS

1. Oto acoustic emission test and BERA test was not done for all cases.
2. The testing was done only once and was not repeated.
3. The BERA test was done only in those cases who failed in OAE test.
4. All infants in Sick neonatal ward have got aminoglycosides the effect of which could not be ruled out.
5. The presence of BERA waves merely tells us that the auditory pathways are normal from the cochlea to the mid brain only. It does not tell us whether the child is actually hearing the sound in the truest sense of the word. Luckily for us the defects in central auditory processing are rather rare as compared to defect in the peripheral auditory pathways.

CONCLUSION

1. Birth Asphyxia can cause hearing impairment in infants.
2. The incidence of hearing impairment is directly proportional to the severity of asphyxia.
3. The incidence of hearing impairment is more in those who required longer duration of inpatient care at sick neonatal ward for control of seizures.
4. The incidence of hearing impairment is higher in those who required assistance during delivery than those who were delivered by labour natural.
5. Screening for hearing impairment is essential in all high risk infants.
6. Hearing impairment is Bilaterally severe in majority of cases (75%).
7. In almost all cases possible site of pathology could be cochlea.

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PROFORMA

The Study of Deafness in term infants with Birth Ashphyxia by oto acoustic emissions and Brain stem evoked response Audiometry tests.

Name :

Age : Sex :

Mother : Father :

Address :

Date of Admission :

Date of discharge :

O/P No. :

Family History :

Consanguinity

Other Siblings

Family History of hearing loss

Antenatal history :

H/o exanthematous fever

H/o drug intake

H/o radiation exposure

Natal & postnatal History :

Mode of delivery Birth weight

Gestational age

H/o Birth Asphyxia

Apgar 1’

5’

H/o Neonatal convulsions

H/o Neonatal hyperbilirubinemia

H/o Hospitalisation

H/o Seizures & Treatment

Developmental History :

Socio Economic History :

General Examination :

Alertness

Neurocutaneous markers

Abnormal Facies

Developmental anomalies

Vitals :

Heart Rate

Respiratory Rate

Weight

Height

Head Circumference

Central Nervous System :

Consciousness

Pupils

Facial Nerve

Response to Sound :

Turning to bell

Startle response

Nasal regurgitation

R L

Tone

UL
LL

Power
UL
LL

Deep Tendon Reflexes

Plantar

Cardio Vascular system

S1 S2

Murmur

Respiratory system

Trachea

Air Entry

Breath sounds

Abdomen

Soft

Organomegaly

Ear, Nose and throat

Head and Neck

Investigation

Hemoglobin

Total count

Differential count

Otoscopy

Otomicroscopy

Electro Encephalogram, Neurosonogram

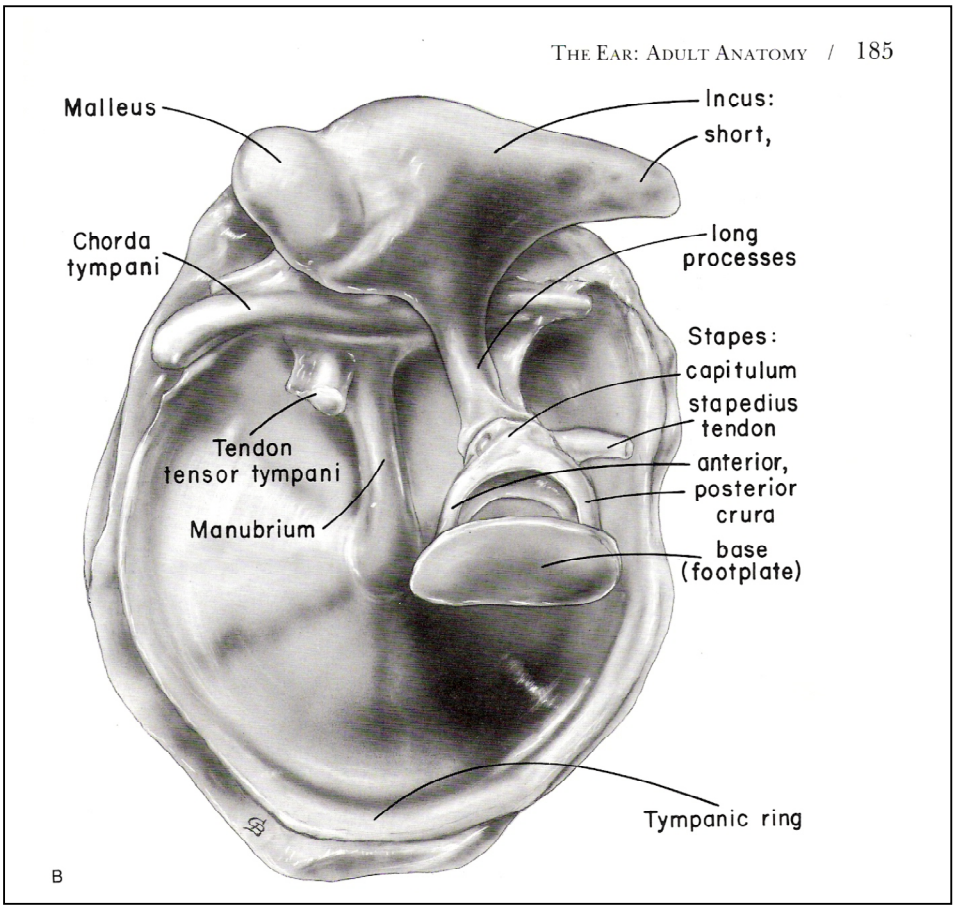
Computed Tomogram Brain

Oto acoustic emissions (OAE)

Brain stem Evoked response audiometry test (BERA)

Inference

TYMPANIC RING AND MEMBRANE –MEDIAL ASPECT



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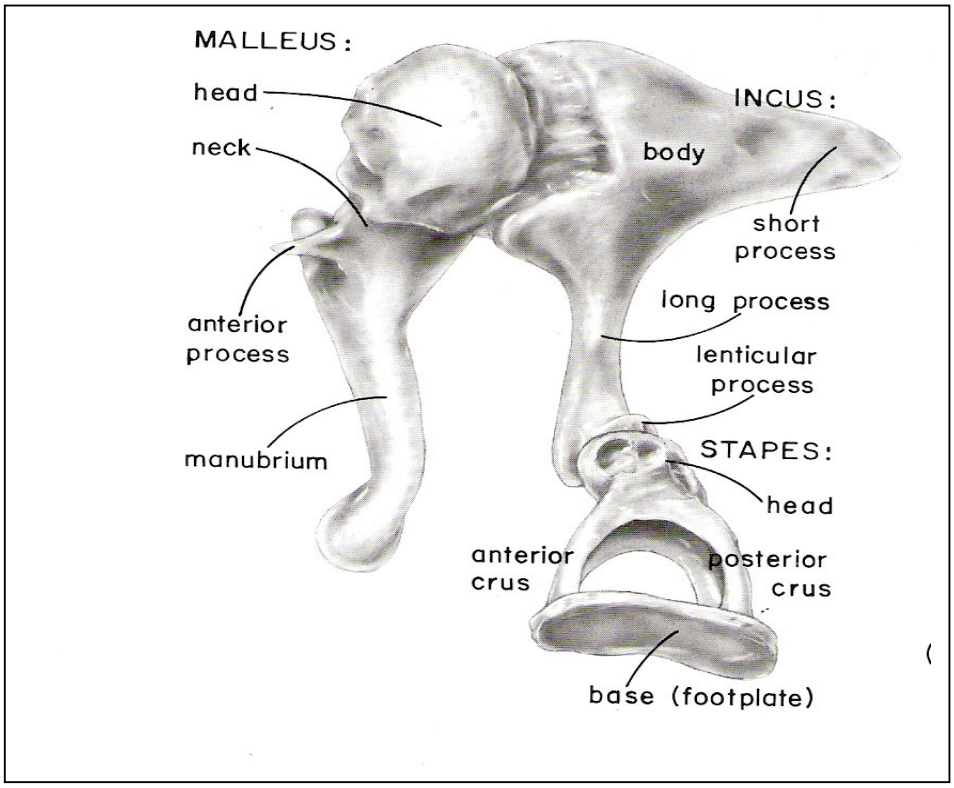

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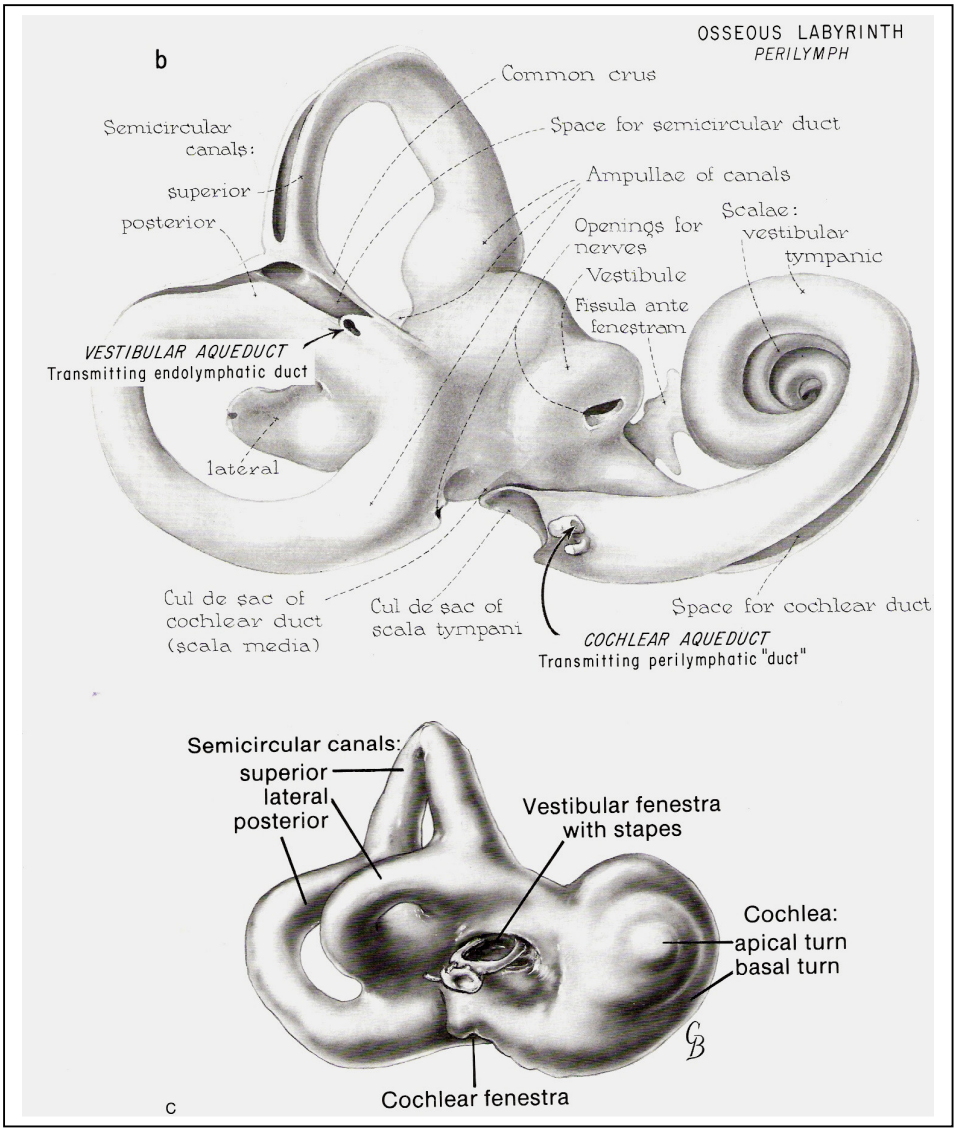
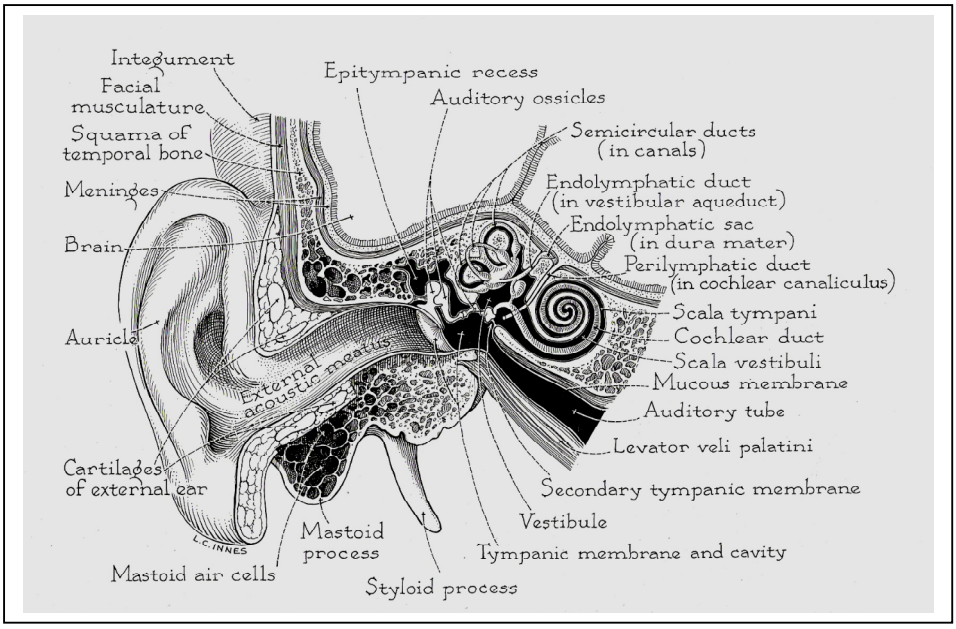
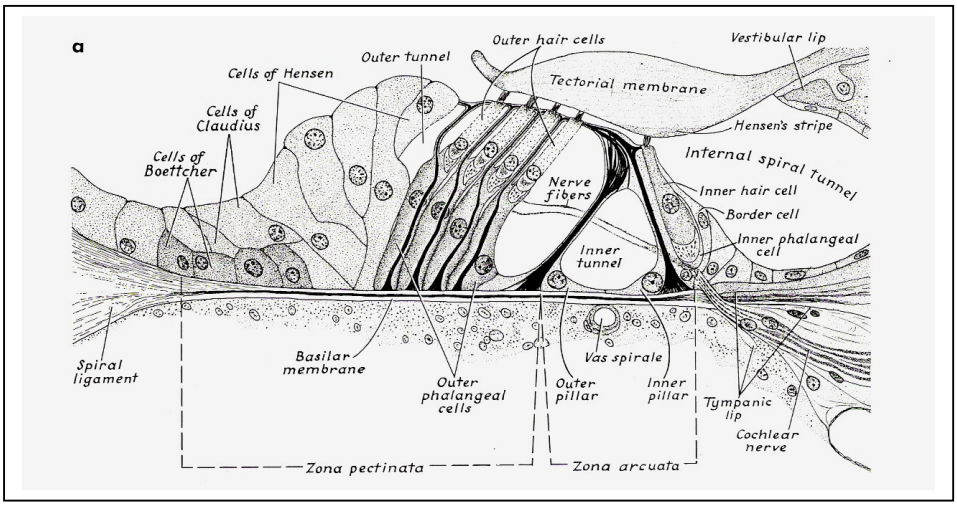


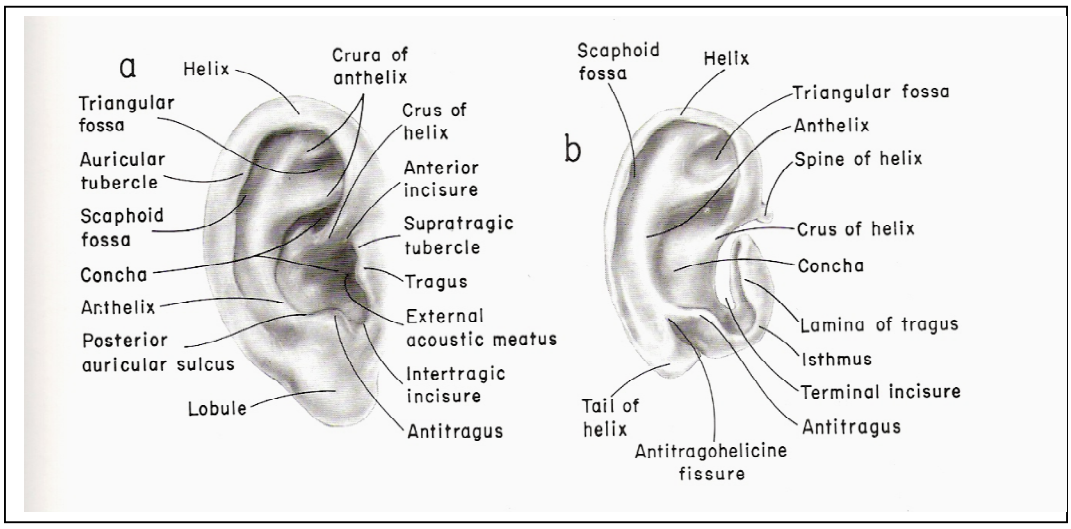
PARTS OF THE EAR



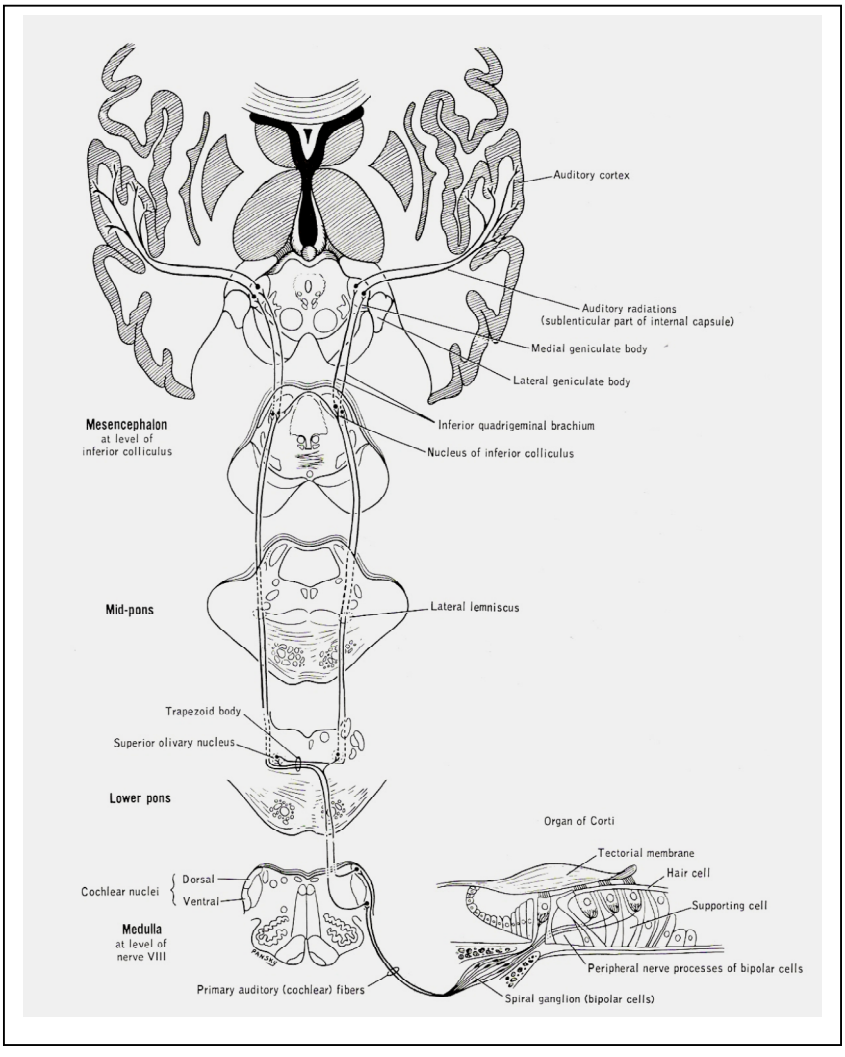
ORGAN OF CORTI



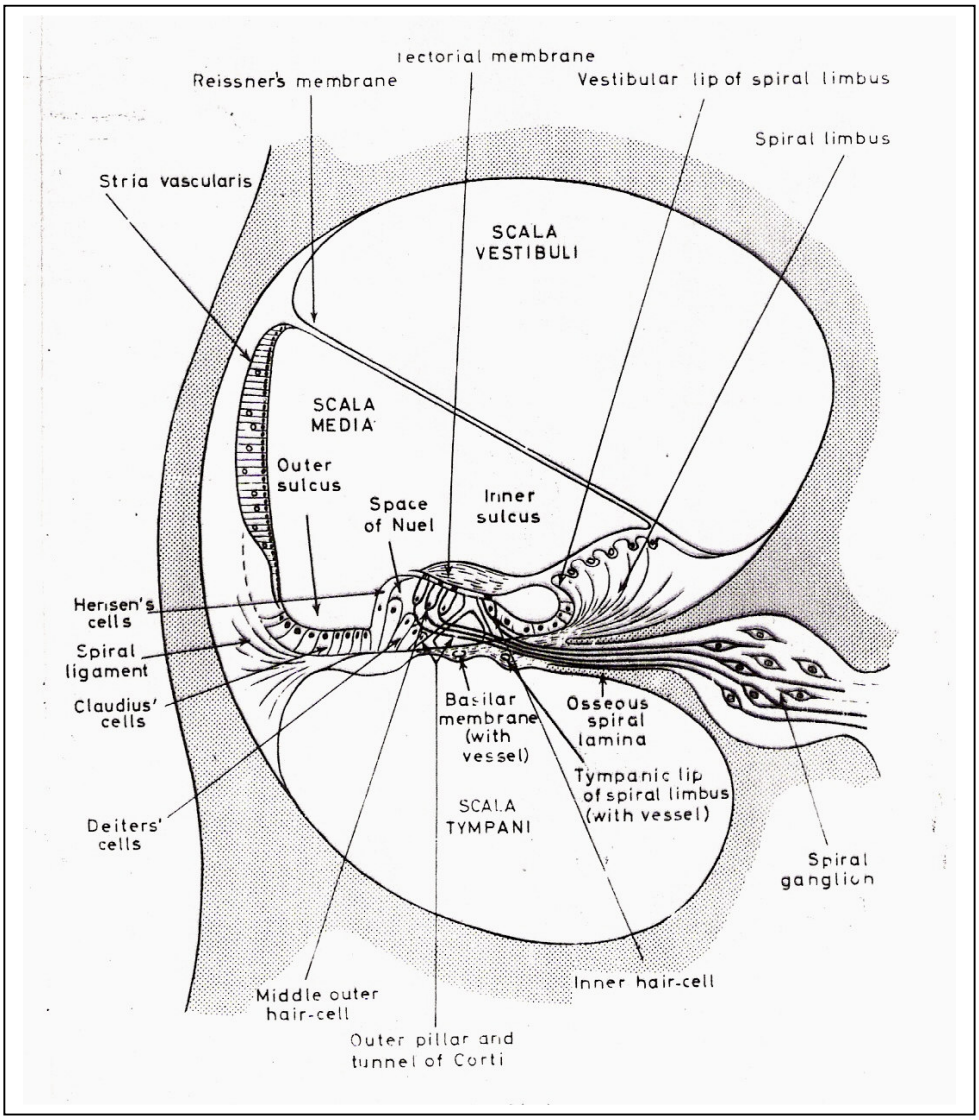
THE PINNA


a
Helix
Crura of anthelix
Triangular fossa
Crus of helix
Auricular tubercle
Anterior incisure
Scaphoid fossa
Supratragic tubercle
Concha
Tragus
Anthelix
External acoustic meatus
Posterior auricular sulcus
Intertragic incisure
Lobule
Antitragus
b
Scaphoid fossa
Helix
Triangular fossa
Anthelix
Spine of helix
Crus of helix
Concha
Lamina of tragus
Isthmus
Terminal incisure
Tail of helix
Antitragus
Antitragohelicine fissure

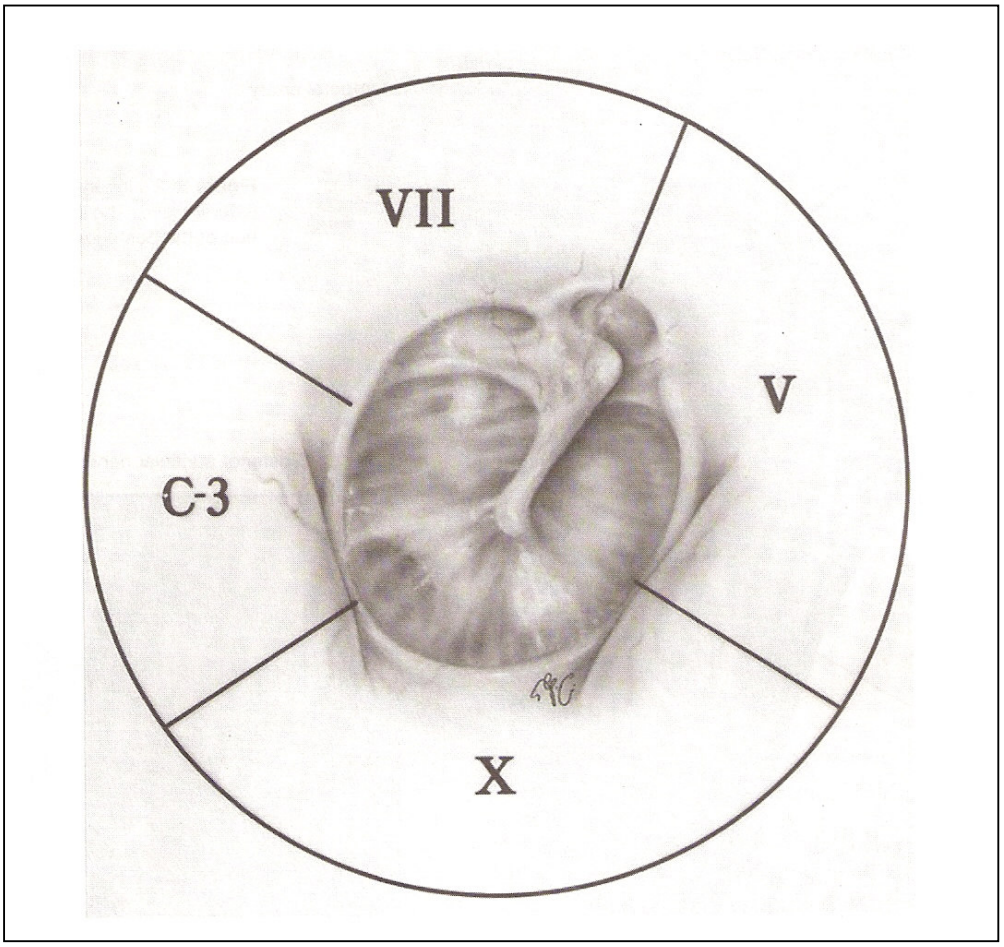
AUDITORY PATHWAY



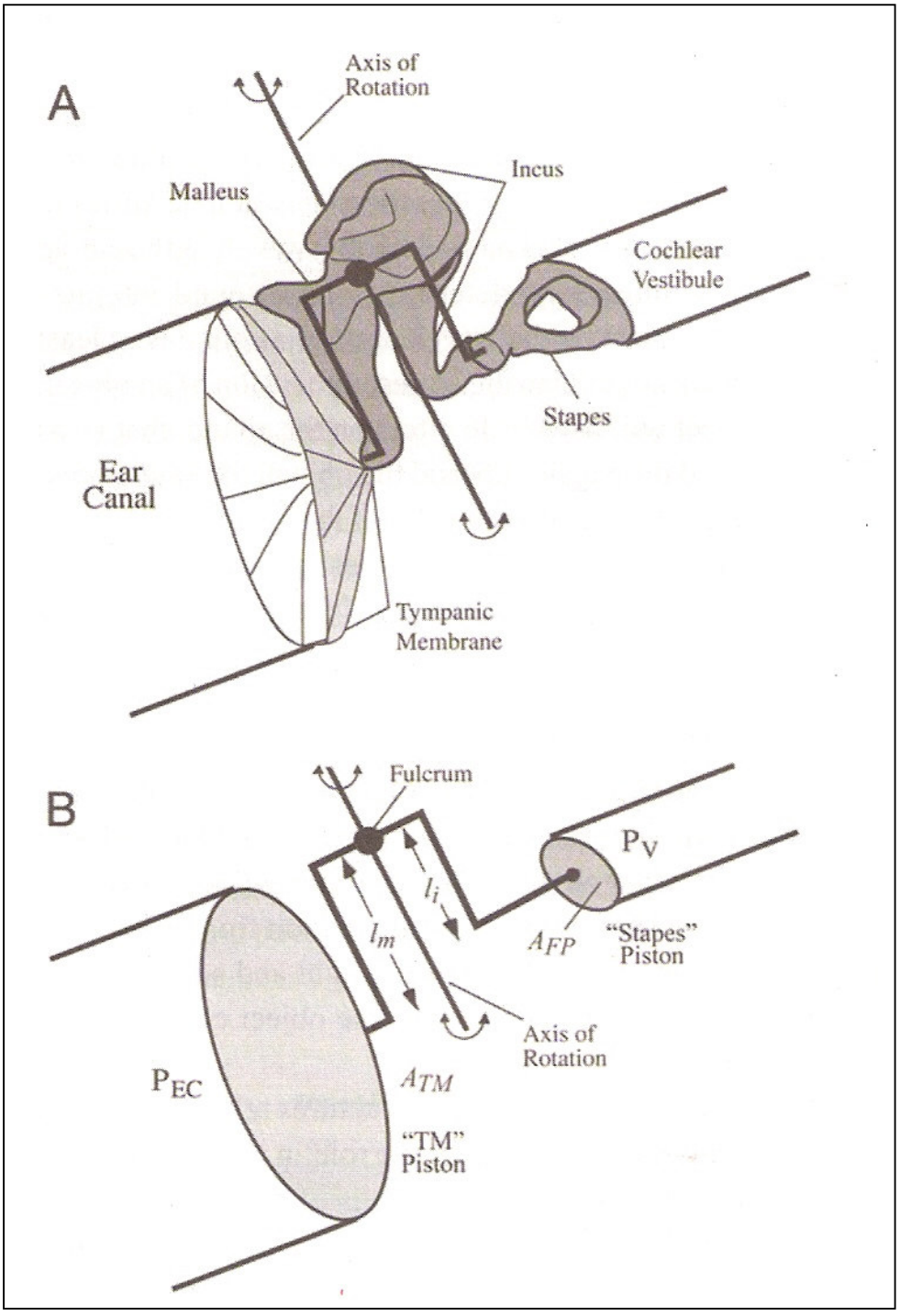
AXIAL SECTION OF COCHLEA



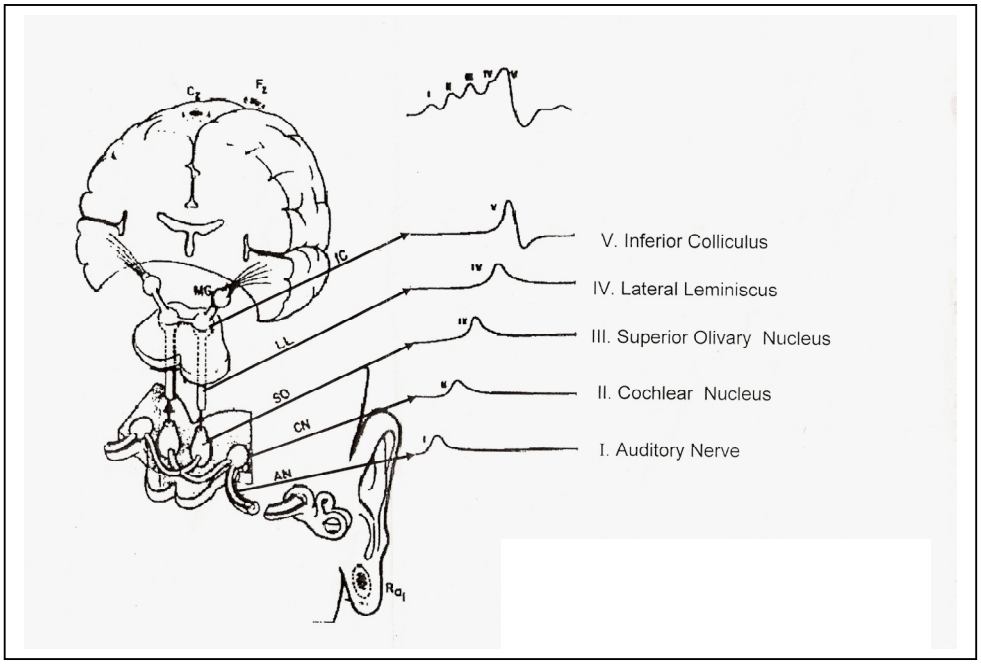
EXTERNAL AUDITORY CANAL – INNERVATION



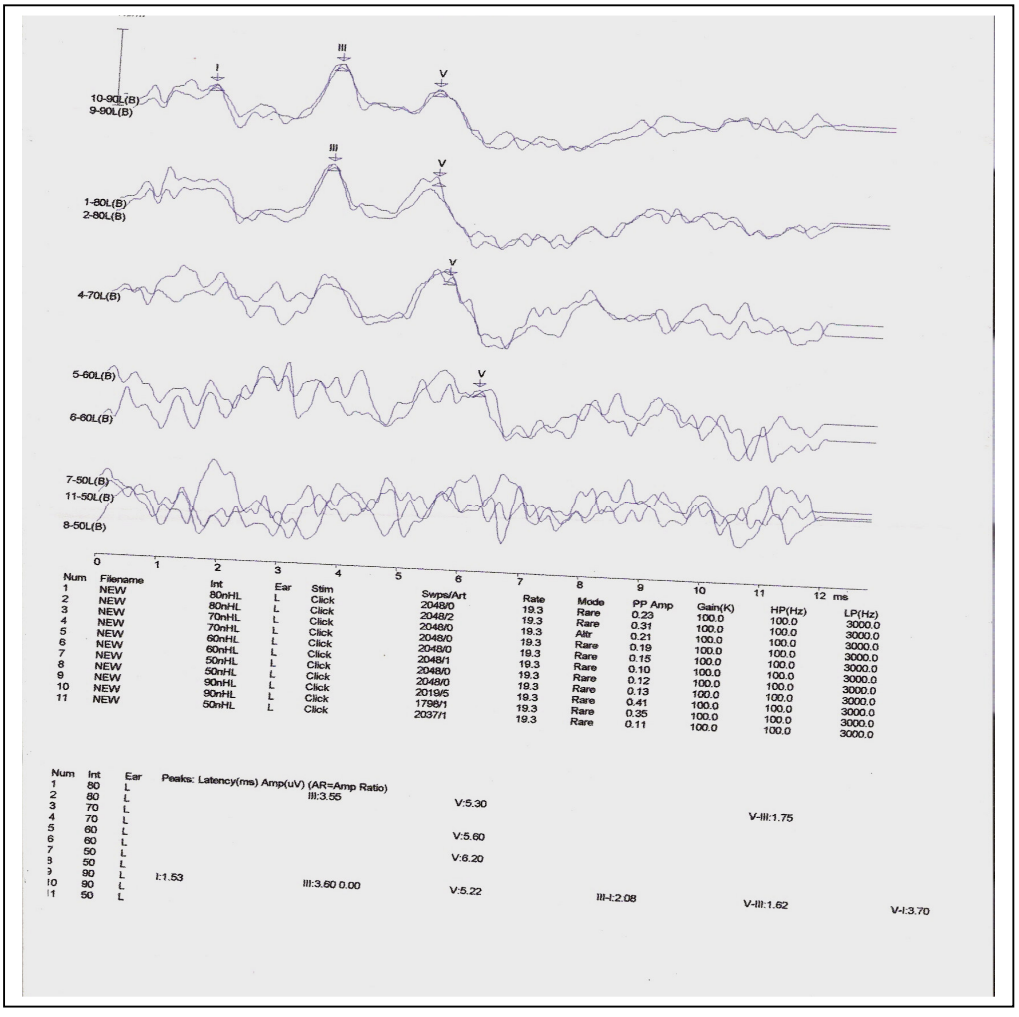
TYMPANO – OSSICULAR SYSTEM



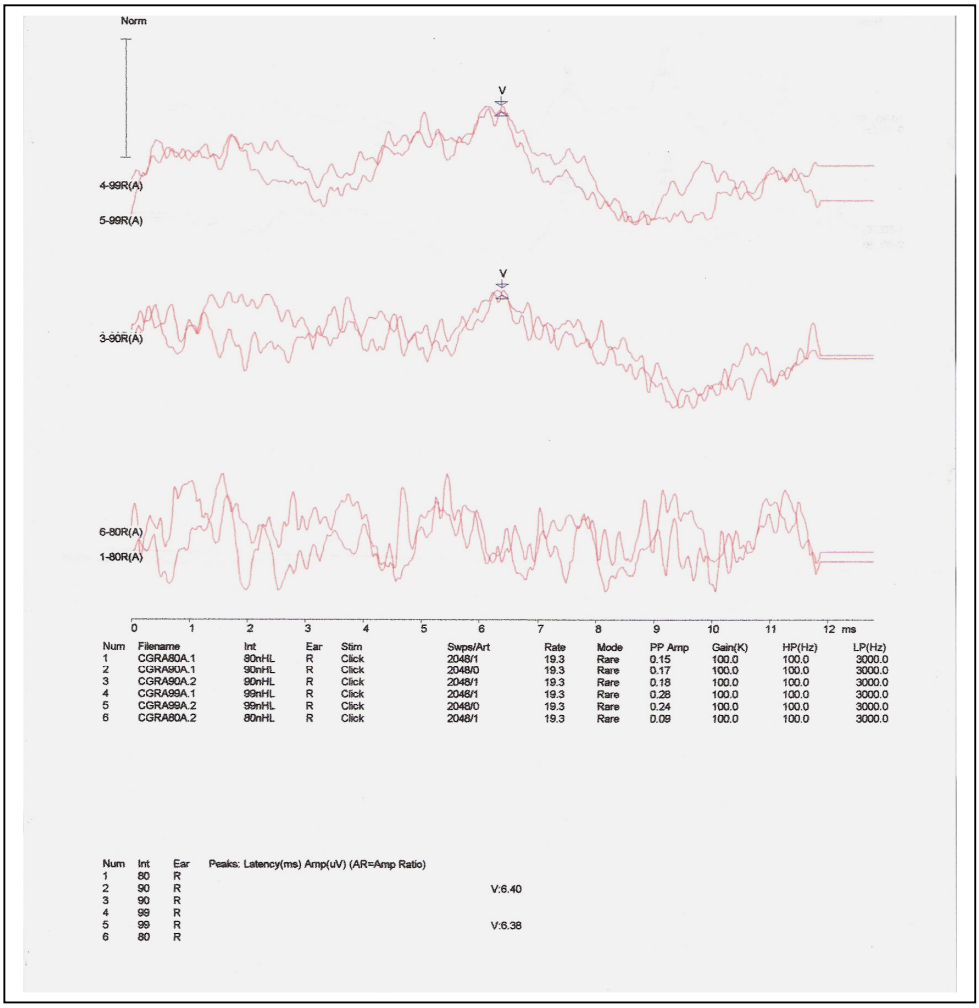
BRAINSTEM AUDITORY EVOKED POTENTIAL



BERA CHART - MODERATE DEAFNESS



BERA CHART – SEVERE DEAFNESS

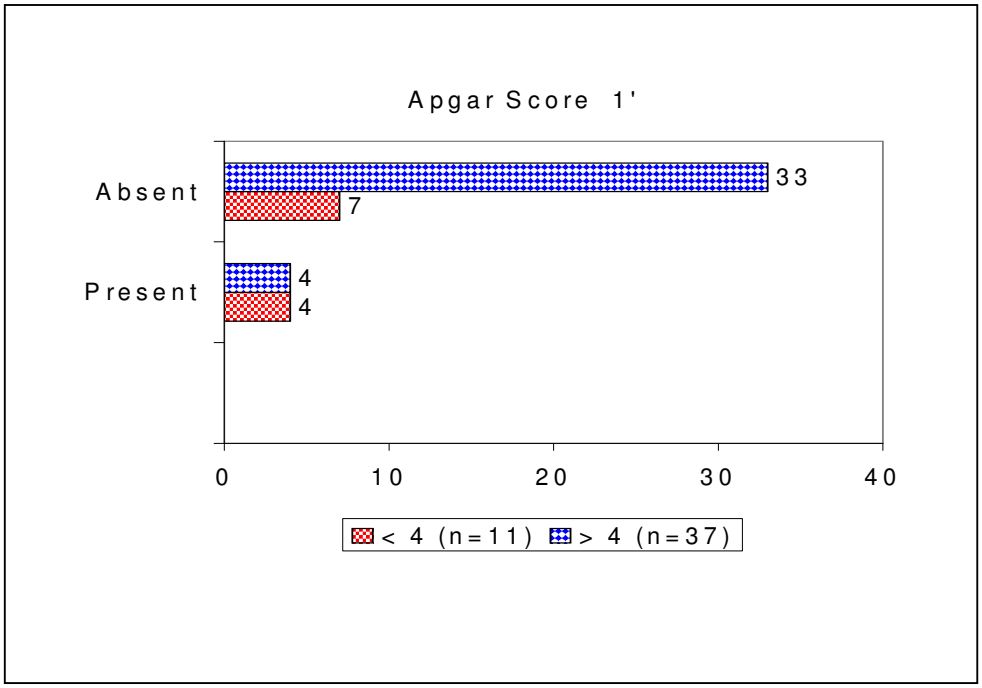


Num	Filename	Int	Ear	Stim	Swps/Art	Rate	Mode	PP Amp	Gain(K)	HP(Hz)	LP(Hz)
1	CGRA80A.1	80nHL	R	Click	2048/1	19.3	Rare	0.15	100.0	100.0	3000.0
2	CGRA90A.1	90nHL	R	Click	2048/0	19.3	Rare	0.17	100.0	100.0	3000.0
3	CGRA90A.2	90nHL	R	Click	2048/1	19.3	Rare	0.18	100.0	100.0	3000.0
4	CGRA99A.1	99nHL	R	Click	2048/1	19.3	Rare	0.28	100.0	100.0	3000.0
5	CGRA99A.2	99nHL	R	Click	2048/0	19.3	Rare	0.24	100.0	100.0	3000.0
6	CGRA80A.2	80nHL	R	Click	2048/1	19.3	Rare	0.09	100.0	100.0	3000.0

Num	Int	Ear	Peaks: Latency(ms) Amp(uV) (AR=Amp Ratio)
1	80	R	
2	90	R	V:6.40
3	90	R	
4	99	R	
5	99	R	V:6.36
6	80	R	

BERA - MACHINE

OAE – MACHINE


Apgar Score 1'
Absent
Present
33
7
4
4
0
10
20
30
40
< 4 (n=11)
> 4 (n=37)

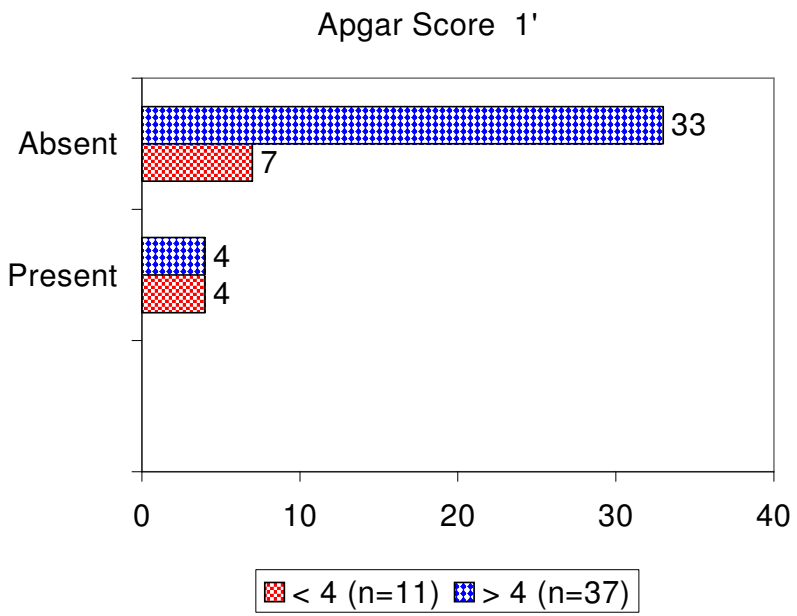
MASTER CHART

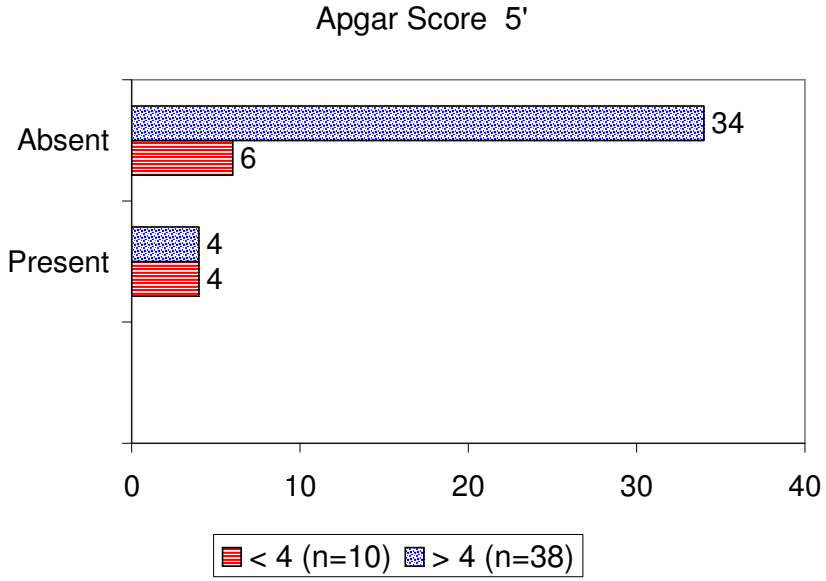
S. No.	Name	OP No.	Sex	BOH	LN / Assisted delivery	B.W	APGAR		Duration of Hospitalisation	Socio economic Status	Neuro Sonogram / CT brain	OAE
							1'	5'				
1.	B/o Jeyanthi	308/05	M	B	A	2.5	3	4	12	V	A	Pass
2.	B/oMariam Beevi	327/05	F	N	LN	3.1	5	6	4	III	N	Pass
3.	B/o Mahababha beevi	332/05	M	B	A	2.5	3	4	8	IV	A	Refer
4.	B/o Prema	356/05	M	N	LN	2.6	5	6	7	IV	N	Pass
5.	B/o Shanthi	360/05	M	N	A	2.3	5	6	8	III	N	Pass
6.	B/o Vijayalaxmi	376/05	M	N	LN	2.8	4	5	4	IV	N	Pass
7.	B/o Shanthi	384/05	F	N	A	2.6	3	3	9	V	N	Refer
8.	B/o Poochendu	401/05	M	N	LN	2.7	5	6	9	V	N	Pass
9.	B/o Syed Ali Fathima	412/05	M	N	LN	2.4	5	6	6	III	N	Pass
10.	B/o Venila	419/05	M	N	LN	2.5	4	5	8	IV	N	Pass
11.	B/o Ramalaxmi	428/05	M	B	A	2.6	4	4	14	V	A	Pass
12.	B/o Raja laxmi	442/05	M	N	LN	2.8	3	3	12	V	N	Refer
13.	B/o Karthigai Rani	463/05	F	N	A	2.9	5	6	7	IV	N	Pass
14.	B/o Rani	466/05	M	N	LN	3.3	4	5	9	V	N	Pass
15.	Rakku	475/05	M	N	LN	2.7	5	6	4	III	N	Pass

16.	B/o Shanthi	505/05	M	N	LN	2.3	5	6	9	V	N	Pass
17.	B/o Muthulaxmi	537/05	M	N	LN	2.8	5	6	8	IV	N	Pass
18.	B/o Muneeswari	553/05	M	N	LN	2.9	4	6	9	V	N	Pass
19.	B/o Chitra	568/05	M	N	A	2.7	3	3	14	V	A	Refer
20.	B/o Jeyanthi	8/05	F	B	A	2.6	3	4	13	V	A	Pass
21.	B/o Ashwarya	18/05	M	N	LN	3.1	6	7	7	V	N	Pass
22.	B/o Laxmi	23/05	F	N	LN	2.9	5	6	12	IV	N	Pass
23.	B/o Samema	29/05	M	B	A	2.4	4	5	14	Iv	N	Pass
24.	B/o Sasikala	38/05	M	N	LN	3.1	6	6	9	III	N	Pass
25.	B/o Thiravium	43/05	F	N	LN	2.5	5	6	9	IV	N	Pass
26.	B/o Puspham	54/05	M	N	LN	2.8	4	5	7	V	N	Pass
27.	B/o Backialaxmi	62/05	M	B	LN	2.8	3	4	13	V	N	Refer
28.	B/o Sakeela	76/05	M	B	A	2.7	5	6	12	V	A	Pass
29.	B/o Mayil	85/05	F	N	LN	2.3	5	6	8	IV	N	Pass
30.	B/o Punitha	93/05	M	N	A	2.6	5	6	8	V	N	Pass
31.	B/o Vijayalaxmi	101/05	M	N	A	3.1	5	5	14	III	N	Pass
32.	B/o Nafeesa	130/05	M	N	LN	2.5	4	5	6	V	N	Pass

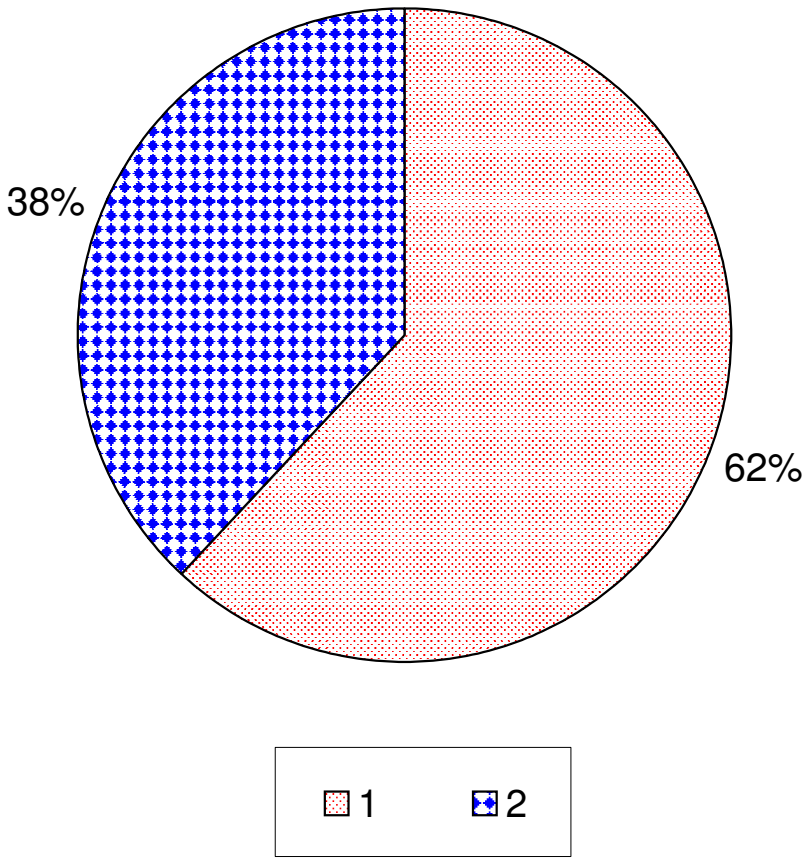
33.	B/o Regina Dhoni	146/05	F	N	LN	2.5	5	6	4	IV	N	Pass
34.	B/o Azhagumeena	155/05	F	N	LN	2.9	3	3	8	V	N	Refer
35.	B/o Deepa	162/05	M	N	LN	2.7	4	6	9	V	N	Pass
36.	B/o Alamely	171/05	M	N	LN	3.2	4	6	8	III	N	Pass
37.	B/o Amutha	178/05	M	N	LN	2.8	5	6	13	V	N	Pass
38.	B/o Laxmi	193/05	M	N	LN	2.9	4	5	8	IV	N	Pass
39.	B/o Mahadevi	209/05	F	B	A	2.5	3	4	7	V	A	Pass
40.	B/o Kurinji malar	220/06	M	B	LN	2.3	5	6	4	V	A	Pass
41.	B/o Paranjothi	225/06	M	N	A	2.5	3	4	12	V	A	Pass
42.	B/o Ananda valli	237/06	M	N	LN	2.5	5	6	6	IV	N	Pass
43.	B/o Suba	250/06	F	N	LN	2.8	4	5	4	III	N	Refer
44.	B/o Panchavarnam	265/06	F	N	A	2.6	3	5	14	V	A	Refer
45.	B/o Nagalaxmi	277/06	M	N	LN	3.2	4	4	9	IV	N	Pass
46.	B/o Kumutha	298/06	F	N	LN	3.1	5	6	8	III	N	Pass
47.	B/o Rekha	317/06	M	B	A	2.8	3	4	13	V	A	Pass
48.	B/o Rajeshwari	349/06	M	N	LN	2.5	4	4	8	V	A	Pass

S. No.	Name	OP No.	Sex	BOH	LN / Assisted delivery	B.W	APGAR		Duration of Hospitalisation	Socio economic Status	Neuro Sonogram / CT brain	OAE
							1’	5’				
3	B/o Mahababha beevi	332/05	M	B	A	2.5	3	4	8	IV	A	Refer
7	B/o Shanthi	384/05	F	N	A	2.6	3	3	9	V	N	Refer
12	B/o Raja laxmi	442/05	M	N	LN	2.8	3	3	12	V	N	Refer
19	B/o Chitra	568/05	M	N	A	2.7	3	3	14	V	A	Refer
27	B/o Backialaxmi	62/05	M	B	LN	2.8	3	4	13	V	N	Refer
34	B/o Azhagumeena	155/05	F	N	LN	2.9	3	3	8	V	N	Refer
43	B/o Suba	250/06	F	N	LN	2.8	4	5	4	III	N	Refer
44	B/o Panchavarnam	265/06	F	N	A	2.6	3	5	14	V	A	Refer

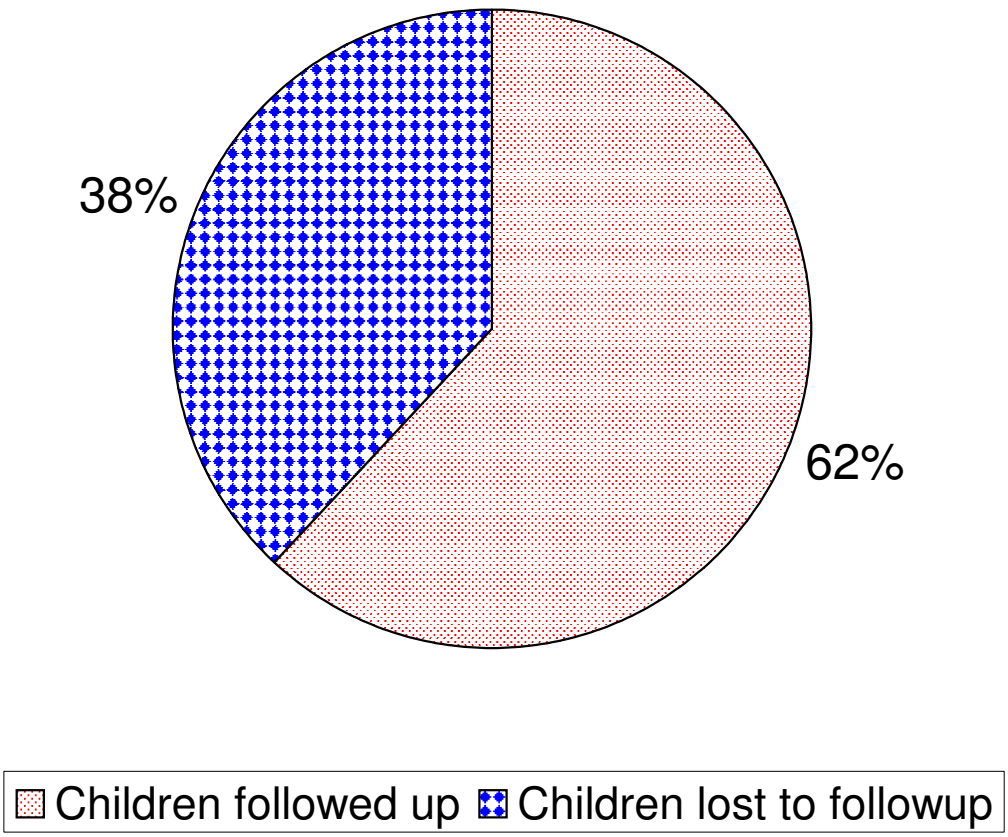

Apgar Score 1'
Absent
Present
33
7
4
4
0
10
20
30
40
< 4 (n=11)
> 4 (n=37)


Apgar Score 5'
Absent
Present
34
6
4
4
0
10
20
30
40
< 4 (n=10)
> 4 (n=38)

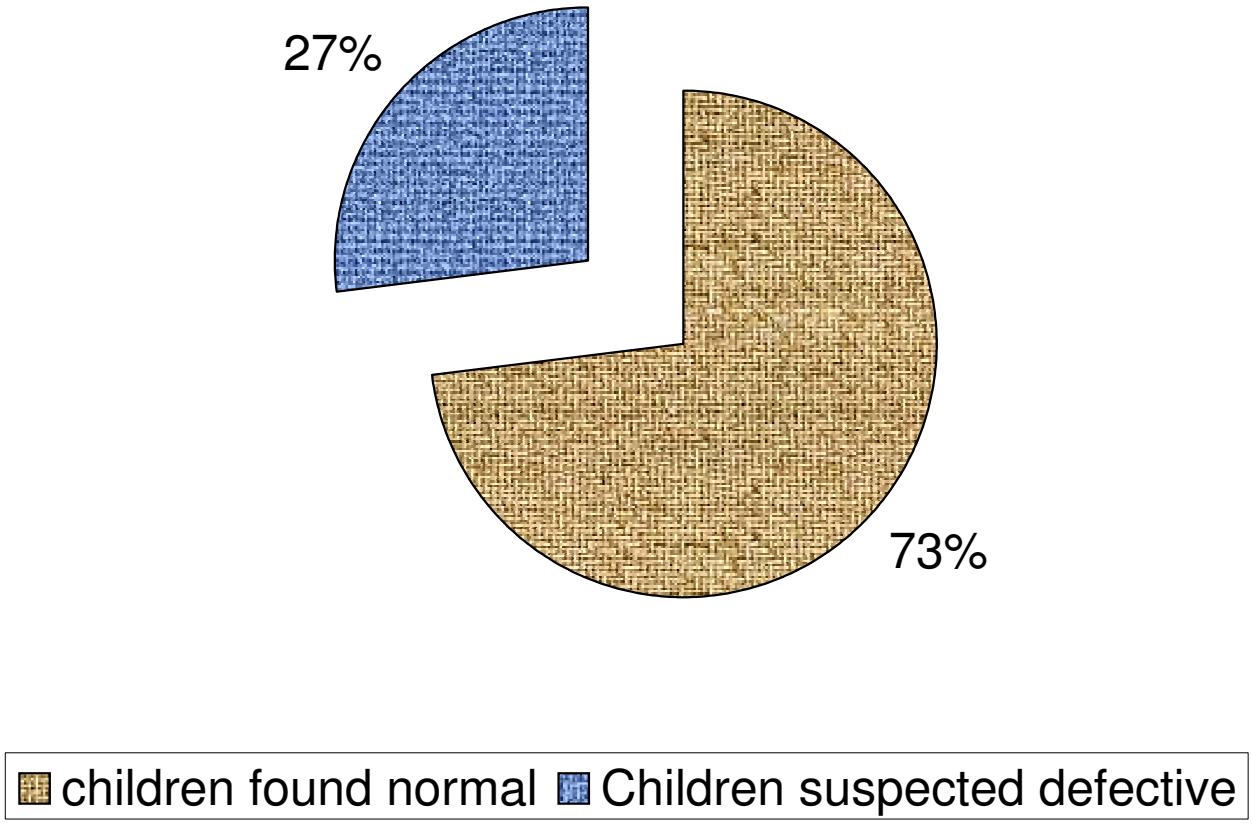
Follow up children enrolled



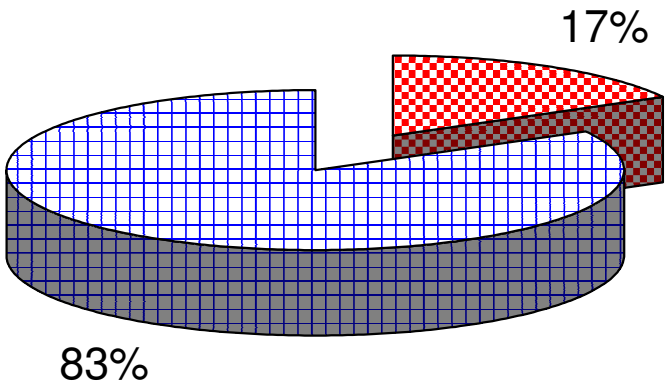
FOLLOW UP OF CHILDREN ENROLLED

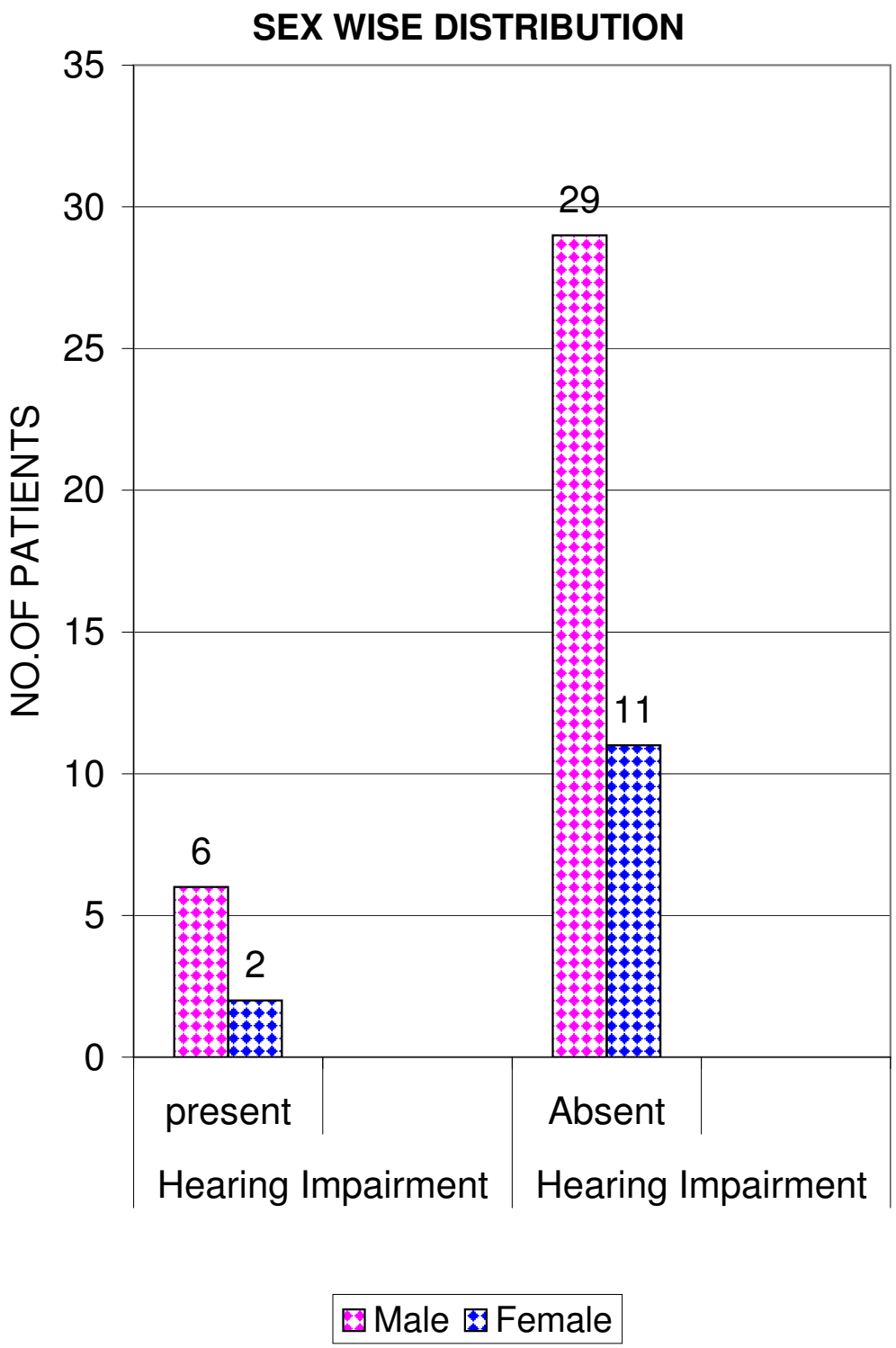


BELL TEST

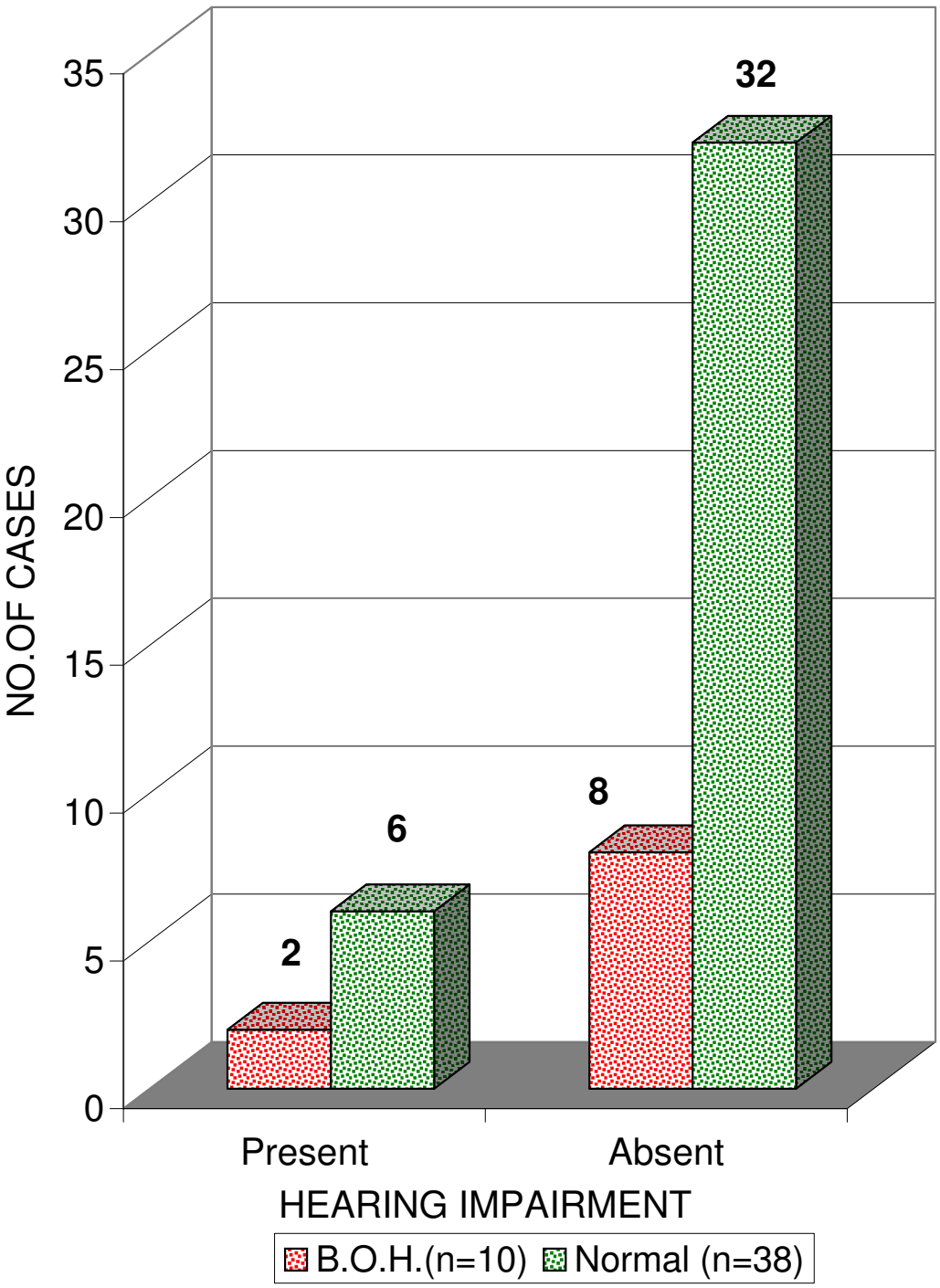


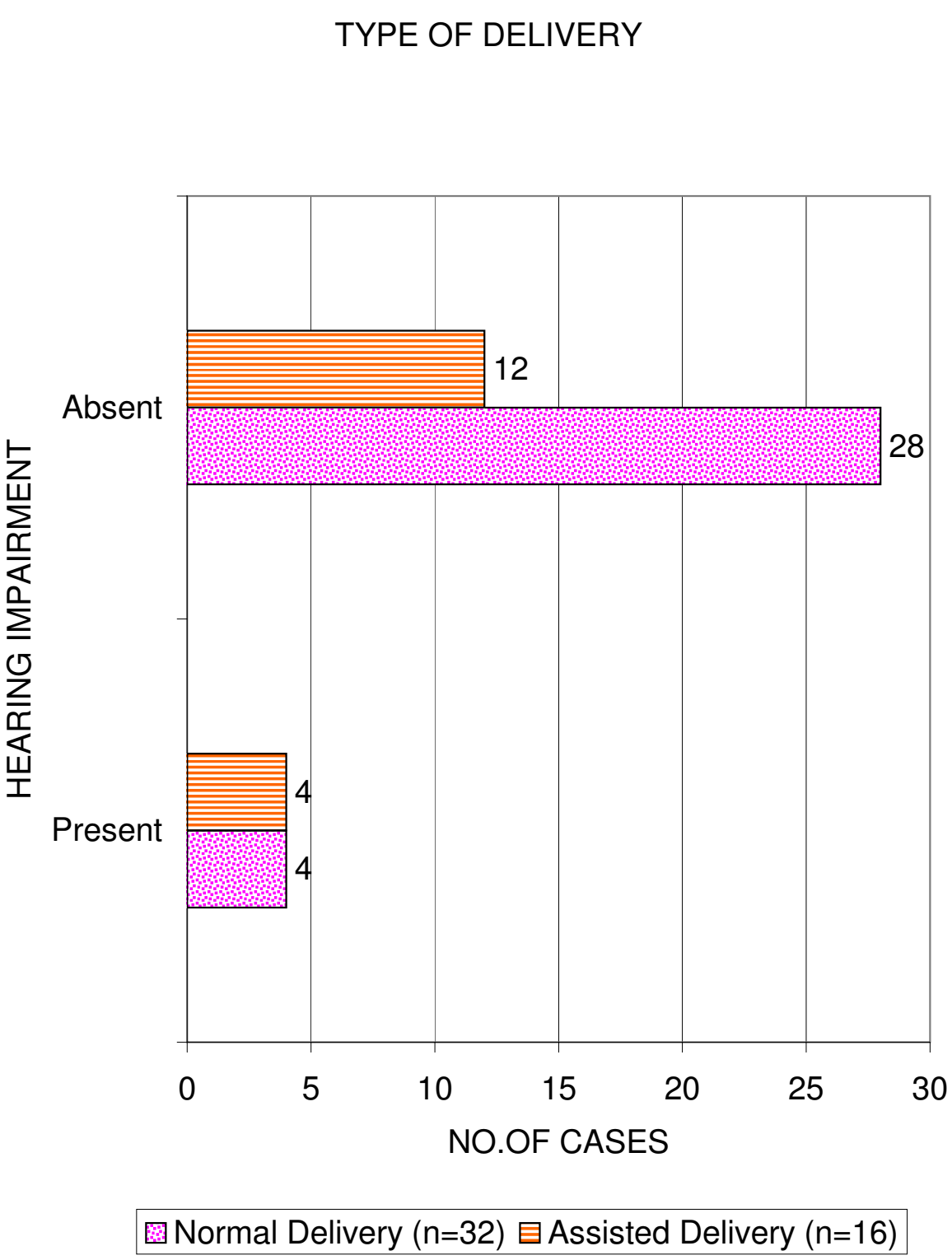
BELL TEST AND OAE TEST



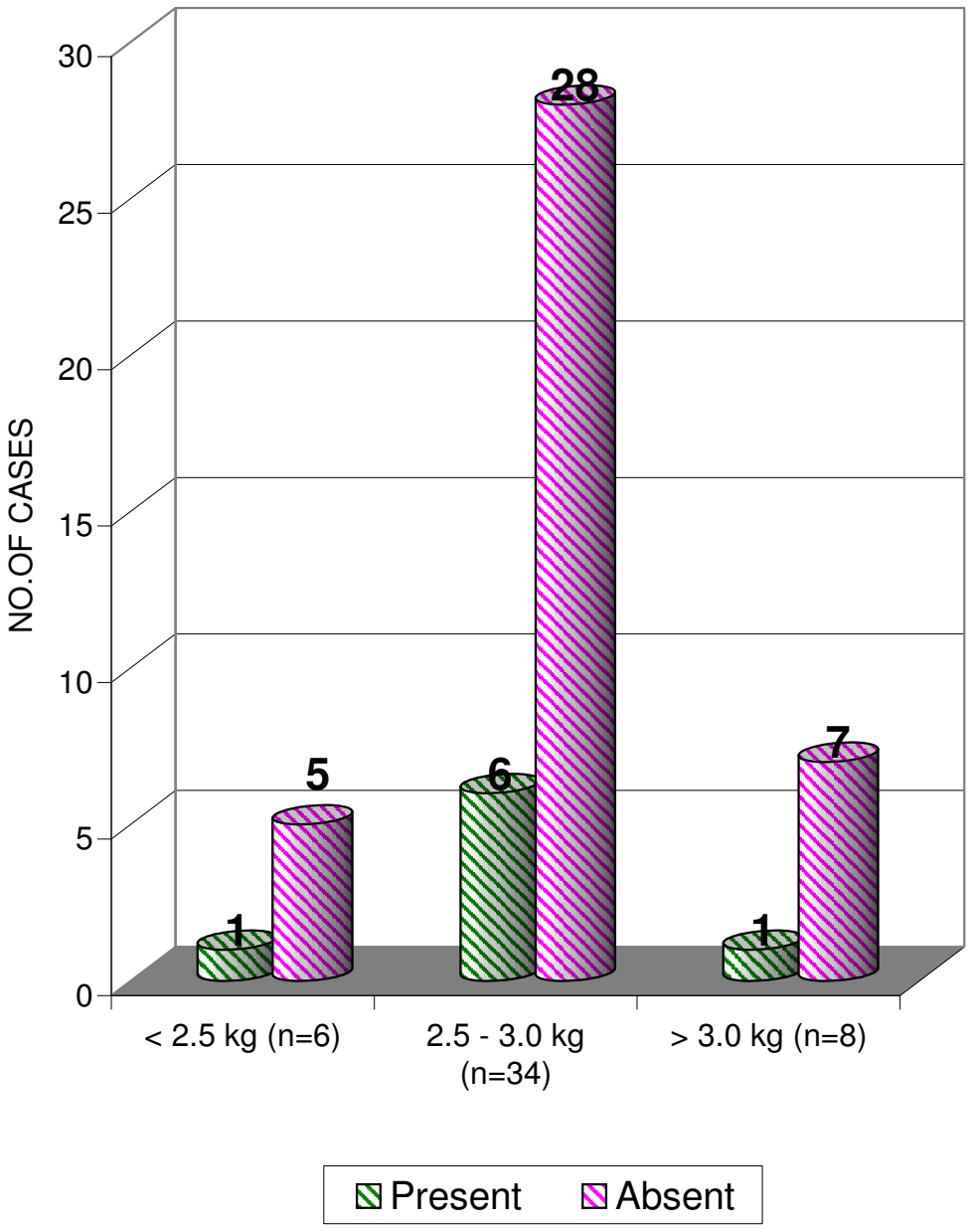

SEX WISE DISTRIBUTION
35
30
25
20
15
10
5
0
NO.OF PATIENTS
29
11
6
2
present
Absent
Hearing Impairment
Hearing Impairment
Male
Female

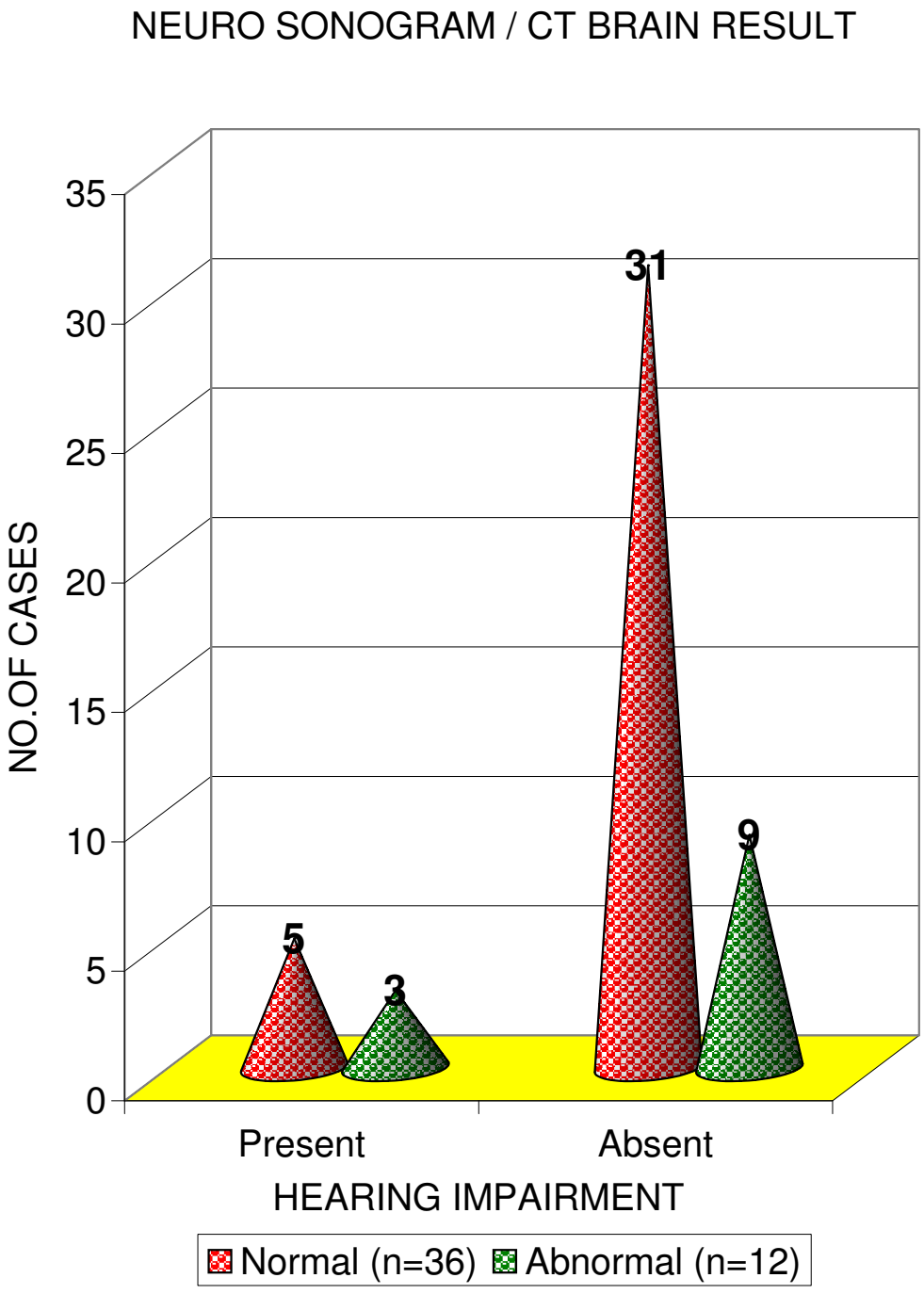
OBSTETRIC HISTORY

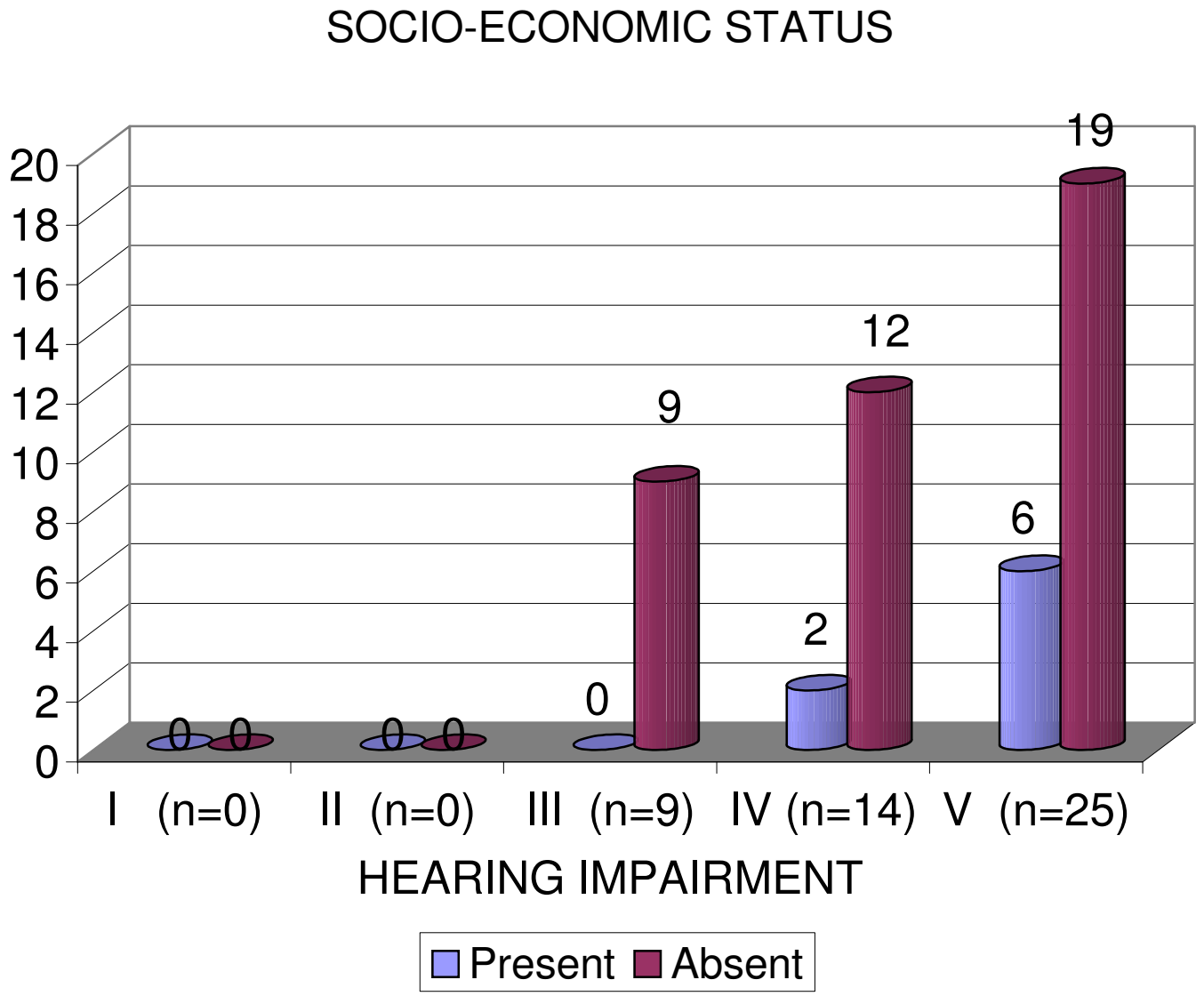


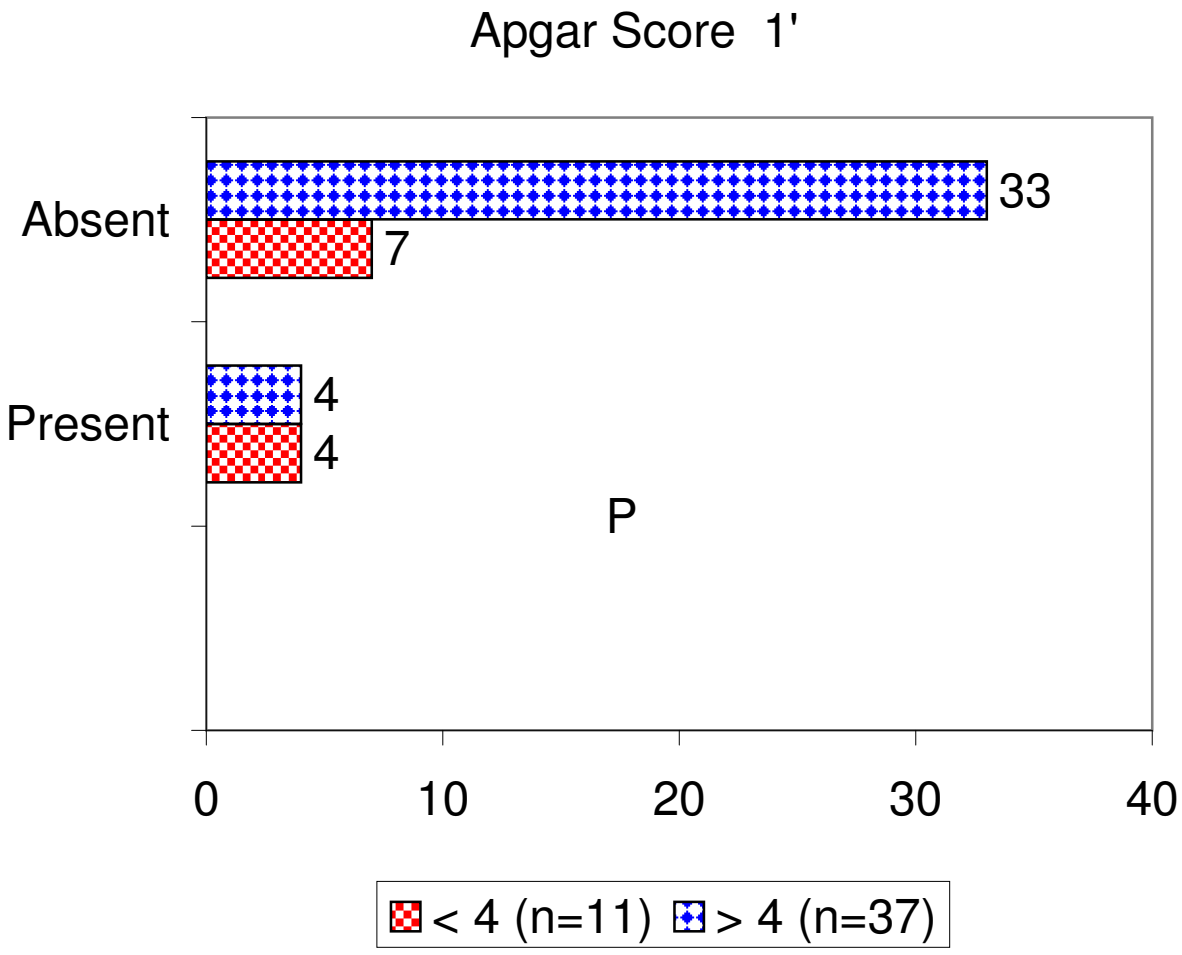

TYPE OF DELIVERY
HEARING IMPAIRMENT
Absent
Present
12
28
4
4
0
5
10
15
20
25
30
NO.OF CASES
Normal Delivery (n=32)
Assisted Delivery (n=16)

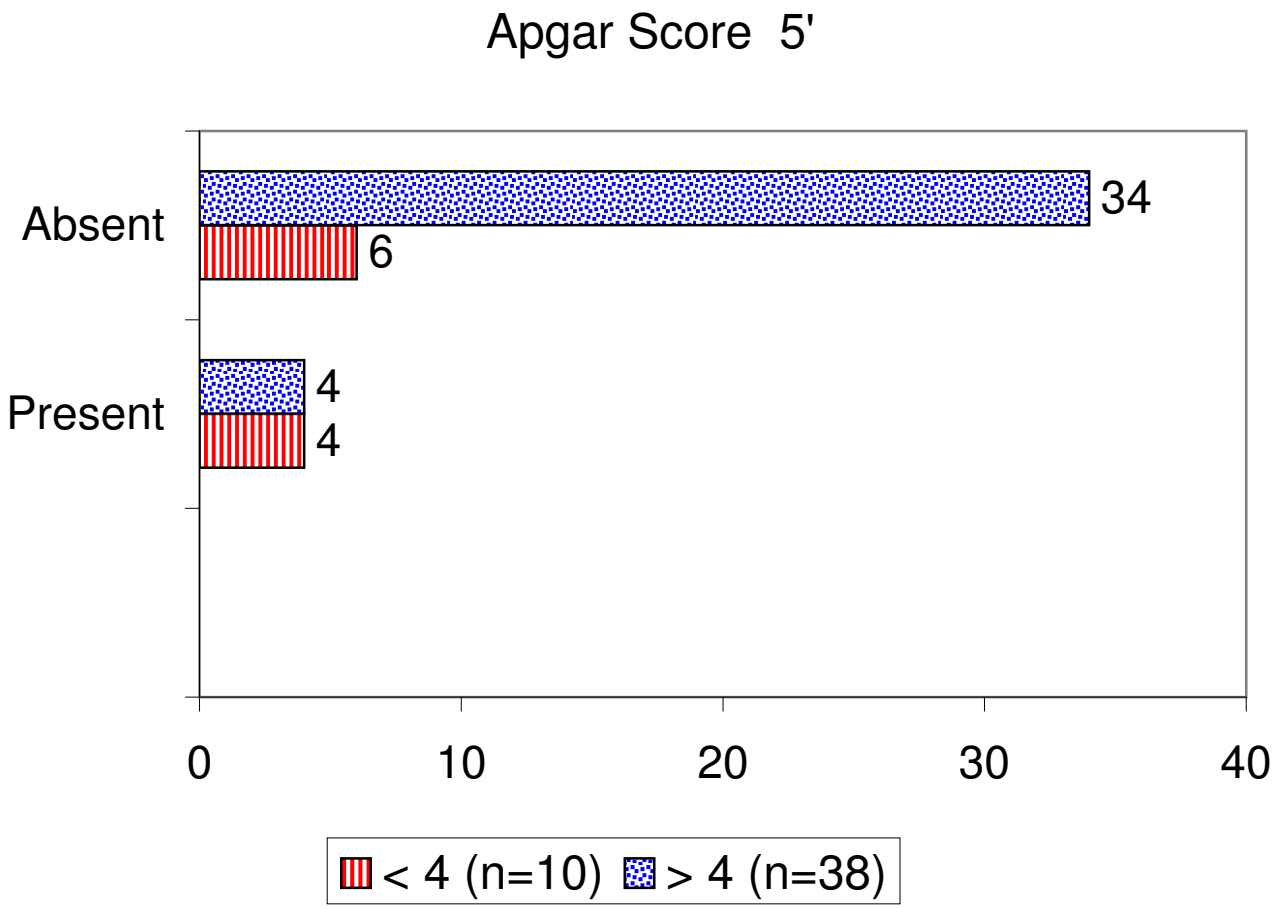
BIRTH WEIGHT




NEURO SONOGRAM / CT BRAIN RESULT
35
30
25
20
15
10
5
0
NO.OF CASES
31
9
5
3
Present
Absent
HEARING IMPAIRMENT
Normal (n=36)
Abnormal (n=12)


SOCIO-ECONOMIC STATUS
20
18
16
14
12
10
8
6
4
2
0
0
0
0
0
9
0
12
2
19
6
I (n=0)
II (n=0)
III (n=9)
IV (n=14)
V (n=25)
HEARING IMPAIRMENT
Present
Absent


Apgar Score 1'
Absent
Present
33
7
4
4
P
0
10
20
30
40
< 4 (n=11)
> 4 (n=37)


Apgar Score 5'
Absent
Present
34
6
4
4
0
10
20
30
40
< 4 (n=10)
> 4 (n=38)

Children followed up	62
Children lost to followup	38

children found normal	73
Children suspected defective	27

Children confirmed defective as per OAE	17
Children confirmed Normal as per OAE	83

Hearing impairment

	Hearing Impairment present	Hearing Impairment Absent
Male	6	29
Female	2	11

	Present	Absent
B.O.H.(n=10)	2	8
Normal (n=38)	6	32

	Present	Absent
Normal Delivery (n=32)	4	28
Assisted Delivery (n=16)	4	12

	Present	Absent
< 2.5 kg (n=6)	1	5
2.5 - 3.0 kg (n=34)	6	28
> 3.0 kg (n=8)	1	7

	Present	Absent
Normal (n=36)	5	31
Abnormal (n=12)	3	9

	Present	Absent
I (n=0)	0	0
II (n=0)	0	0
III (n=9)	0	9
IV (n=14)	2	12
V (n=25)	6	19

Apgar score	Present	Absent
< 4 (n=11)	4	7
> 4 (n=37)	4	33

Apgar score	Present	Absent
< 4 (n=10)	4	6
> 4 (n=38)	4	34